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CEREBRO-SPINAL MENINGITIS.[1]

By W. L. CALOV, M.B., Ch.M., D.T.M. (Sydney), F.R.A.C.P.,

Honorary Relieving Assistant Physician, Sydney Hospital; Honorary Physician, The Anti-Tuberculosis Association of New South Wales; Honorary Assistant Physician, The Royal South Sydney Hospital.

THERE are some of us who do not know a great deal about cerebro-spinal meningitis. It may be that before this year is out we shall need all the knowledge that we can muster. Hence this paper. I am not vain enough to regard this as an important contribution to medical science. I can only hope that it will refresh the minds of some and help others to a clearer understanding of this remarkable disease, as the compiling of it has helped me.

SYNONYMS.

Cerebro-spinal meningitis is also known as cerebro-spinal fever, epidemic cerebro-spinal meningitis, epidemic cerebro-spinal fever, meningococcal meningitis, malignant purpuric fever, spotted fever, petechial fever, black death (Ireland), *méningite cérébrospinale* (France) and *Genickstarre* (Germany). The disease known as posterior basal meningitis is usually meningococcal. Inflammation of the meninges of the brain and cord may be caused by organisms other than the meningococcus; therefore the name "cerebro-spinal meningitis" is not a particularly happy one. "Cerebro-spinal fever", though it is used perhaps more than any of the other names, is scarcely sensible. "Meningococcal meningitis" is probably the best in use at present. Perhaps "meningococcal fever" would be even better; but I have not the courage to use it nor to adopt the American "meningococcal meningitis", good as I believe it to be.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 30, 1940.

DEFINITION.

Cerebro-spinal meningitis is an infectious disease, occurring sporadically, or in epidemics in which the incidence in relation to the population is low; it is caused by the meningococcus, and characterized by inflammation of the meninges of the brain and spinal cord, by great irregularity of its clinical course and by a high mortality rate.

HISTORY.

Vieusseux described an epidemic of cerebro-spinal meningitis at Geneva in 1805. This is the earliest account on record.[1] The first known appearance of the disease in the United States of America was in 1806, when Danielson and Mann described "a singular and very mortal disease" in Massachusetts.[2] There were occasional epidemics in the United States of America and Europe throughout the nineteenth century. Osler[3] states that "while soldiers are peculiarly liable, cerebro-spinal fever has never been a great war pestilence"; up to the time of the Great War there were 62 epidemics in France, 43 of which were confined to troops. There is no reference to the disease in accounts of the Napoleonic (1790-1815), Crimean (1854-1856), Italian (1848-1866) and Danish (1848-1850) wars.

There were a few cases only in the Franco-Prussian War (1870-1871), mainly in the environs of Paris. Isolated cases occurred in the Boer War (1899-1902) and the Russo-Japanese War (1904-1905). The only exception in the nineteenth century was the American Civil War (1861-1865), both armies being affected; but no epidemic was widespread. The disease broke out in epidemic form in 1893 in three mining towns in Maryland, United States of America. It died down after a time and reappeared in an outbreak in Baltimore and Washington in 1898. It prevailed in a mild form in 27 States and broke out again in epidemic form in New York in 1904. During 1904-1905 there were 6,755 cases and 3,455 deaths from cerebro-spinal meningitis in New York, while Philadelphia, less than 100 miles away, was free. Texas was the scene of an epidemic in 1912. The commencement of mobilization of troops in America in 1917 heralded a widespread epidemic, which was not put down until the end of the war.

Epidemics occurred in Glasgow in 1907 and Belfast in 1907-1908. There was no extensive epidemic in England or Wales until the winter of 1914-1915. During the war years it was prevalent throughout England, soldiers in barracks and camps being peculiarly liable. Cases also occurred among British troops on the Western Front. The numbers are shown in Table I.[4] There was no serious outbreak in any other theatre of war. Among French and German troops the incidence was lower than among British.

TABLE I.

Incidence of Cerebro-Spinal Meningitis in the British Expeditionary Force in France during the Years 1915–1918.

Year.	Cases.	Incidence per Thousand of Strength.	Deaths.	Mortality Percentage.
1915	313	0·55	—	—
1916	393	0·33	138	35·1
1917	701	0·43	198	28·2
1918	176	0·11	69	39·2

Cerebro-spinal meningitis has been recognized in Australia since the beginning of the present century, if not longer; but no serious epidemic occurred until the winter of 1915, the first winter after the declaration of war.

The following figures show the numbers of cases reported to the New Zealand health authorities from 1916 to 1920.

Year.	Number of Cases.
1916	135
1917	42
1918	159
1919	96
1920	79

Since the Great War there have been numerous minor outbreaks in gaols and institutions, so that a more or less stable incidence seems to have been maintained in most countries. In England and Wales the incidence was greatly increased during the years 1931, 1932 (Yorkshire epidemic) and 1933, as the following figures show.

Year.	Number of Cases.	
1927	470	Civilians only
1928	412	
1929	650	
1930	664	
1931	2,157	Figures include non-civilians
1932	2,136	
1933	1,695	

The first winter after the declaration of war has again been associated with a great increase in the number of cases in England, Scotland and Wales (see Table II and Figure I), though not in Eire (a neutral country) or northern Ireland,[5] where there are probably no very large military encampments. It is of interest to note that the number of cases reported to the health authorities during February and March alone is higher than the average annual number during the years 1914-1918, notwithstanding Captain Balfour's statement in the House of Commons that the incidence "is no greater than can be expected in peace-time".[16]

TABLE II.

The Incidence of Cerebro-Spinal Meningitis in England, Scotland and Wales from October 7, 1939, to March 9, 1940, compared with the Incidence in the Corresponding Week of the Previous Year.

Period.	Number of Cases.	Number of Cases during Corresponding Week of Previous Year.
Week ended—		
October 7, 1939	27	18
October 14, 1939	28	20
October 21, 1939	25	21
October 28, 1939	31	23
November 4, 1939	24	20
November 11, 1939	29	25
November 18, 1939	47	25
November 25, 1939	33	30
December 2, 1939	39	23
December 9, 1939	33	38
December 16, 1939	30	31
December 23, 1939	32	22
December 30, 1939	61	30
January 6, 1940	70	27
January 13, 1940	130	58
January 20, 1940	178	50
January 27, 1940	236	45
February 3, 1940	370	46
February 10, 1940	498	44
February 17, 1940	674	62
February 24, 1940	720	49
March 2, 1940	738	61
March 9, 1940	711	58

ÆTIOLOGY.

The Causal Organism.

Cerebro-spinal meningitis is caused by the meningococcus, the *Diplococcus intracellularis meningitidis* of Weichselbaum, which was first described by Leichtenstern in about 1887. During the Great War the role of the meningococcus was questioned by Hart,[6] who expressed the opinion that the disease was caused by a filter-passing virus. He claimed to have proved that filtered cerebro-spinal fluid in two cases of cerebro-spinal meningitis contained a virus that was highly infective to monkeys. But this is of historical interest only.

War.

During the Great War of 1914-1918 mighty armies were mobilized with amazing rapidity; hundreds of thousands of recruits, many of whom were unaccustomed to hardship, strain or violent physical

exercise, were crowded together in inadequate quarters and subjected to intensive training. It was in the barracks and training camps that the epidemics were bred. In France and Germany, in addition to the large standing armies, all the men eligible for service had been trained before the outbreak of hostilities. During the war the only men requiring training were recruits, who were

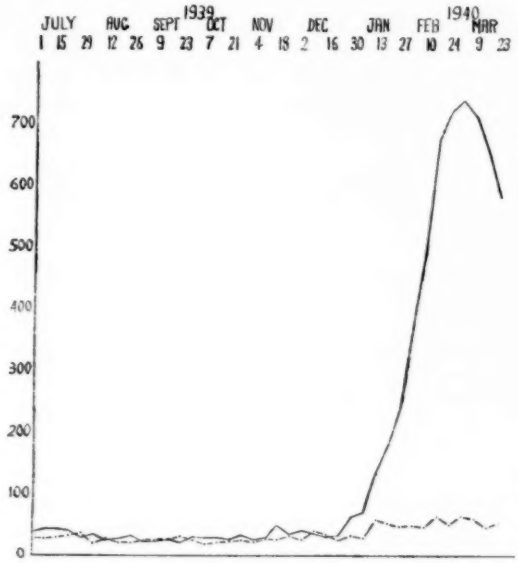

FIGURE I.

Graph showing the incidence of cerebro-spinal meningitis as reported week by week in England, Scotland and Wales, from July 1, 1939, to March 23, 1940, compared with the incidence at corresponding periods twelve months earlier.

called up as they reached military age. In those countries there was not the same need for the concentration of large bodies of raw recruits. This seems to be one reason why the incidence of cerebro-spinal fever was high in the British and American armies and not in the Continental.

A further factor was that the military population was essentially unstable. Detachments of men recruited from widely separated parts were constantly marching into camp, and bodies of trained men were constantly being sent to the war zones or to other camps. This constant shifting of the population made possible the contact of far greater numbers with infected persons than would have been possible in a more or less fixed community.

The incidence among soldiers was higher than among the general population.[4][8] The relative incidence among soldiers and civilians in the Victorian epidemic of 1915 is shown in Figure II. A notable feature is the quick fall in the military rate, which occurred, presumably, because of the greater control over the soldiers.

In the United Kingdom during the years 1914-1918 there were 10,259 cases, 4,238 of which occurred in the army. The navy was not severely affected. It was found that most of the cases in the navy occurred among men who had recently entered the service.[9] Fildes and Baker[9] express the opinion that recruits ("new entries" in the navy) are more susceptible because (i) many of them come from distant sparsely populated parts, to be crowded with city dwellers and infected with, to them, unfamiliar bacteria; (ii) many of them are in poor physical condition. But these are probably of less importance than some other factors, such as: fatigue from unaccustomed exertion; lowering of resistance from inoculation, vaccination and strange food; overcrowding (*vide infra*) and exposure.

A further possibility is that the virulence of the infecting organisms becomes enhanced by passage when large numbers of susceptible persons are crowded together. Murray[14] has observed an increase in virulence as a result of passage through laboratory animals.

The epidemic in the civil population during the Great War probably arose in the military camps. There was free contact between soldiers and civilians, and soldiers on leave were scattered throughout the country, conveying infection to every community. There are records of a number of small outbreaks that had their origin in the visit of infected soldiers.[8]

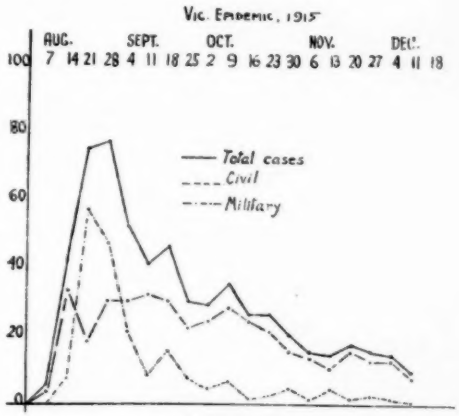

FIGURE II.

Showing the relative incidence of cerebro-spinal meningitis in the army and in the civil population in the Victorian epidemic of 1915. Note the rapid fall of incidence in the army, presumably due to more adequate control. (Copied from Fairley and Stewart,[8] after the Departmental Medical Committee's Report, Victoria, 1915.)

Naso-Pharyngeal Inflammation.

Inflammation of the naso-pharynx appears to be of some importance in the spread of the infection, although doubt has been expressed whether the meningococcus itself ever causes pharyngitis. Andrewes, Bulloch and Hewlett[10] write:

> While it cannot be denied, in face of some of the observations, that individual cases of cerebro-spinal fever may be ushered in by sore throat, the more usual relation between the disease and catarrh seems a fortuitous one dependent on the fact that catarrh and cerebro-spinal fever have a similar seasonal prevalence: there is the same sort of relation as there is between catarrh and the price of coal.

On the other hand, many authorities, including Lundie, Thomas and Fleming,[11] and Herrick,[12] claim to recognize three separate stages of cerebro-spinal meningitis, the first being "the catarrhal stage".

Naso-pharyngeal catarrh is important because it causes coughing and sneezing. Colebrook and Tanner, working on behalf of the Medical Research Committee,[10] found that no meningococci grew on plates of culture medium held directly under the nostrils of a carrier breathing heavily for several minutes; but the organisms grew as a result of "explosive expiration" over a plate, sneezing on a plate produced many colonies, and in two of five tests coughing on plates held vertically a foot away from the subject resulted in a culture of meningococci. The exposure of plates at the heads of beds or on tables in a room occupied by seven carriers resulted in a growth of the organisms in only one of five experiments.

In the United Kingdom this year the influenza wave has been higher than usual, but not so much higher as to account for the great increase in the incidence of cerebro-spinal meningitis. In Figure III the numbers of cases of cerebro-spinal meningitis

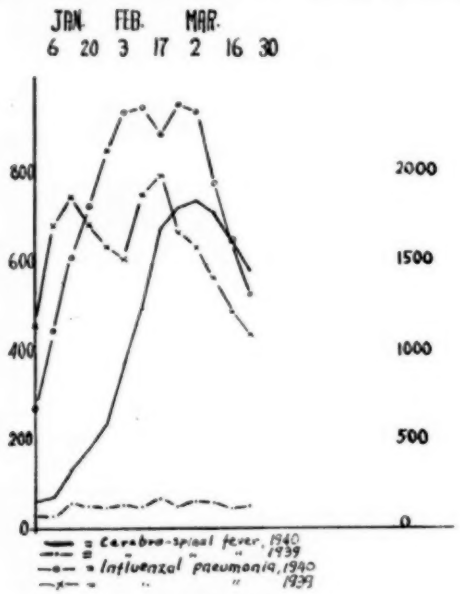

FIGURE III.

Graph showing the incidence of pneumonia (influenzal only in Scotland; both influenzal and primary in England and Wales) and cerebro-spinal meningitis in 1930 and 1940 in England, Scotland and Wales. The incidence of pneumonia, which may be taken as an index of the incidence of respiratory infection, is higher in 1940, but not sufficiently higher to account for the great increase in the incidence of cerebro-spinal meningitis.

for the first few months of this year are plotted against the numbers of cases of pneumonia, including influenzal pneumonia, for the same period and the numbers of cases of cerebro-spinal meningitis and pneumonia, including influenzal pneumonia, for the corresponding periods of 1939. The incidence of pneumonia, side by side with that of cerebro-spinal meningitis in a normal year (1939), is shown in Figure IV.

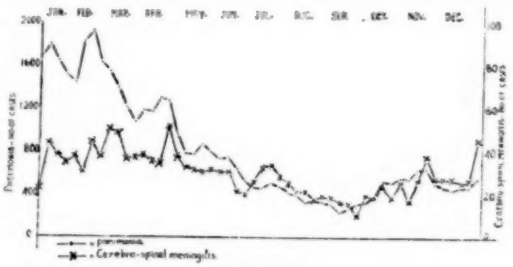

FIGURE IV.

The incidence of influenzal pneumonia in Scotland and influenzal and primary pneumonia in England and Wales compared with the incidence of cerebro-spinal meningitis during 1939.

Carriers.

There are always persons who harbour meningococci in the naso-pharynx. Some of them are temporarily infected, having recently come in contact with another carrier or a patient suffering from cerebro-spinal meningitis; some harbour the organism for months. In normal times carriers are rare; but preceding and during an epidemic the carrier rate in the affected community may be as high as 70% or even higher. Glover[13] concludes that a rate of 2% to 5% "may be considered usual under the best conditions obtainable in barracks and hutments". But it may be taken that in war-time as well as in peace the rate in the civil population is lower than this, excepting just before or during an epidemic. Glover states that a carrier rate of 20% should be regarded as a danger signal. He states:

> A wave of high (non-contact) carrier rates *precedes* and accompanies an outbreak of cerebrospinal fever.
>
> In other words there is a carrier epidemic (for the most part entirely devoid of symptoms) *preceding* and accompanying the much smaller case epidemic.
>
> The meningeal cases are the visible foam on the dark carrier rate wave; when the latter reaches a certain height (20 per cent.) something happens, and cases appear, just as when a water wave reaches a certain height the surface tension cracks, and it becomes a visible "white horse".
>
> Conditions other than height of wave may modify the appearance of the foam, but a certain height is essential.

Sporadic cases may occur in any community where there are carriers; but no epidemic occurs without this preliminary rise in the carrier rate.

According to Glover, the reasons are: (i) the higher the carrier rate, the greater the chance a susceptible person has of sleeping next to a carrier, (ii) a higher carrier rate means overcrowding, which means lowered resistance, (iii) the higher the carrier rate, the more abundant and more nearly pure are the cultures obtained from pharyngeal swabbing, (iv) in a community with 33% of carriers, every third man is a carrier of a dangerous type.

> Add to increase the carrier's range and frequency of discharge the explosive sneeze of an influenza, which has also lowered the resistance of the susceptible recruit,

already temporarily decreased by recent inoculation and vaccinia, by the new hardship and by nostalgia, and we have the exact conditions requisite for an outbreak.

Glover found that there were 100 carriers to every one who contracted the disease. The average resistance is high, and to overcome it massive dosage seems to be necessary.

It is of some importance that carriers themselves may become affected with cerebro-spinal meningitis, perhaps after they have harboured the organisms for many weeks. This is a rare occurrence, however; Fildes and Baker[9] did not observe one case among 485 carriers in the navy during 1916 and 1917.

Overcrowding.

Overcrowding is the most important single factor in the development of an epidemic of cerebro-spinal meningitis. Overcrowding in a military camp or barracks causes an increase in the carrier rate and a higher proportion of persons in the camp infected with virulent organisms. The carriers of virulent organisms infect groups of civilians, and so arises a civilian as well as a military epidemic.

Overcrowded military establishments in time of war must be regarded as the main source of all widespread epidemics.

Glover[13] found that when the regulation army beds, two feet six inches wide, were three feet apart, the carrier rate was less than 2%. The closer the beds were to one another, the higher was the rate. If beds were separated by less than nine inches the carrier rate was 28% to 30% (see Figures V and VI). It did not seem to matter how much

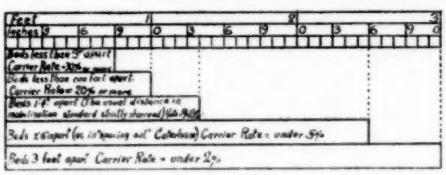

FIGURE V.

Showing the results of crowding and of spacing beds on the carrier rate at Caterham Depot during the Great War. (After Glover.)

cubic space was allotted to each man. Infection depends on a man's being within range of blasts of droplets from his neighbour's naso-pharynx.

Glover did not conduct any properly controlled investigation of the relation of overcrowding to carrier rate in bell tents; but he makes some interesting observations. When there are seven men to a tent, heads are five feet apart, a condition similar to that in a barrack room when beds are spaced at intervals of two feet six inches. There should never be more than seven men to a tent. If there are fifteen men in a tent, the centres of their heads are two feet four inches apart, and if two men lie facing each other their mouths are not more than one foot eight inches apart, "a condition obviously very favourable to infection of a respiratory nature". He remarks incidentally that: "The pressure of feet at the pole with 15 or 16 men is so great that the wearing of boots is almost essential to sleep."

It should be noted that overcrowding has to be in existence for three weeks to produce the great rise in carrier rate noted by Glover.

While it is necessary to stress the great importance of overcrowding, it must not be overlooked that people may live in conditions of herd-like crowding and almost unimaginable squalor and yet

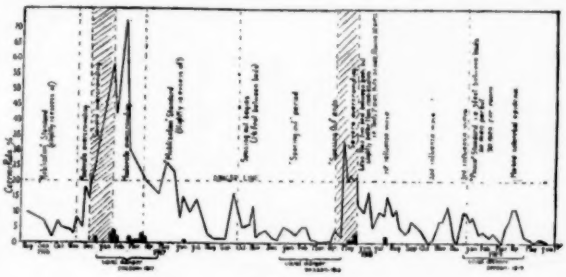

FIGURE VI.

Graph showing the harmful effects of overcrowding and the beneficial effects of spacing beds at Caterham Depot during the Great War. (After Glover.[13])

not be peculiarly liable to cerebro-spinal meningitis, even when an epidemic is occurring in the vicinity. This statement demands qualification. In times of peace the inmates of institutions and soldiers in barracks are the people most liable to the disease. It seldom breaks the bounds of the institution or barracks. In India, for example, epidemics have occurred in gaols without spreading to the presumably law-abiding section of the population,[15] although the prisoners were cleaner, better fed, better clothed and better housed, and had more room than the people outside. The obvious reason is that prisoners are unable to carry infection beyond the confines of their prison. But the question may be asked: "Why are the prisoners more liable to the disease?" The answer, in part at any rate, is that in the gaol large numbers of people live in continuously close contact, providing conditions most suitable for a rise in the carrier rate, whereas in the general population there is crowding of the members of individual families or of individual households, but not of large bodies of people. Furthermore, the inmates of gaols are gathered from widely separated places; they may be regarded as samples of individual communities; one or more of them may come from a community where there is a high percentage of carriers of a virulent strain of the meningococcus. If such a strain were introduced into a crowded household it would cause an outbreak in that household but not necessarily in the rest of the community.

Inadequacy of Ventilation.

Much of the value of protection against overcrowding may be lost if ventilation is inadequate. It seems scarcely possible that the authorities in Great Britain have forgotten the lessons taught by Glover in the Great War; yet during the past few months cerebro-spinal fever has raged there in

epidemic form. Difficulties in ventilation have been increased by the necessity for the nocturnal "black-out". It may be that while crowding has been minimized ventilation has been neglected.

Season.

In cold and temperate climates most cases occur in late winter and early spring: in the northern hemisphere, from February to April; in Australia, from August to October (see Figures VII, VIII and IX). In the tropics these rules do not apply.

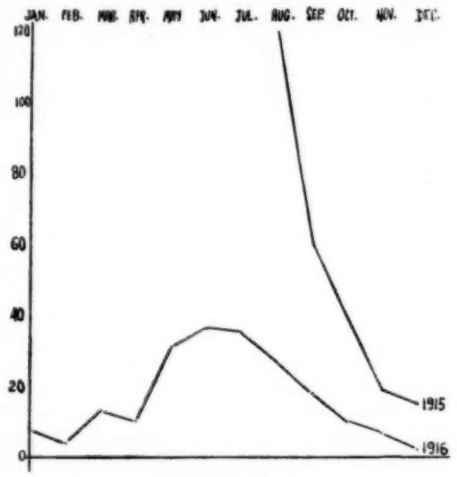

FIGURE VII.

Graph showing the seasonal incidence of cerebro-spinal meningitis gauged by the numbers of patients admitted to the Alfred Hospital, Melbourne, in 1915 and 1916.[6]

Buchanan[15] states that in India most of the cases in his experience occurred in April, in the dry season, when dust was plentiful. Corkill[17] states that in the Northern Sudan, epidemics develop in the dry season and finish in the wet, but tend to

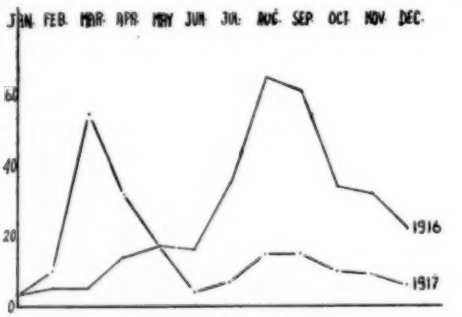

FIGURE VIII.

Graph showing the seasonal incidence in New South Wales in 1916 and 1917. A remarkable feature is the high peak in March, 1917.

diminish before the end of the dry season and even before there is any pronounced increase in humidity. He says that the decline of an epidemic commences when the sky becomes cloudy. He believes that vitamin *A* deficiency is responsible for the susceptibility of the inhabitants of Northern Sudan. The excessive ultra-violet irradiation resulting from the continuous bright sunshine of the dry season "activates the deficiency state", causing increased susceptibility. Clouds diminish the ultra-violet radiation; hence the decline of an epidemic on the approach of the wet season. He suggests that winter vitamin *A* deficiency and spring sunshine might be largely responsible for European epidemics.

A simpler explanation is that winter and spring are the seasons of colds and influenza, hence coughing and sneezing and the readier transmission of naso-pharyngeal infection.

FIGURE IX.

Graph showing the seasonal incidence in New South Wales as gauged by the dates of admission of 103 patients to the Royal Prince Alfred Hospital from 1910 to 1937. The effect of the abnormal March curve of 1917 (see Figure VIII) is seen.

Age and Sex.

Cerebro-spinal meningitis is essentially a disease of youth. Infants and children are the most susceptible. Persons over thirty years of age are less susceptible than younger people. Of 102 patients admitted to the Royal Prince Alfred Hospital from 1910 to 1937, only 13 were aged more than thirty years. The actual incidence (approximately the percentage incidence) according to age was as follows:

Age.	Number of Cases.	
0- 5 years	18	26
6-10 years	8	
11-15 years	15	35
16-20 years	20	
21-25 years	16	28
26-30 years	12	
31-35 years	7	9
36-40 years	2	
42 years	1	
51 years	1	
52 years	1	
62 years	1	

In this series the proportion in the age group 16-30 is probably higher than would be shown by an analysis of figures for the whole State, because large numbers of young soldiers were admitted to the Royal Prince Alfred Hospital during 1915, 1916 and 1917, when the epidemic in the army was at its height. Figures given by Fairley and Stewart[8] are set out in Table III. It will be seen from this that 29·9% of the patients in the Victorian epidemic of 1915, as compared with 26% of the Royal Prince Alfred Hospital patients, were ten years of age or less; 24·2%, as compared with

35%, were aged eleven to twenty years, and 26·8%, as compared with 28%, were aged twenty-one to thirty years. In the Victorian figures the influence of army life is seen in the great preponderance of young adult males over females in the same age groups.

TABLE III.

The Ages and Sex of 628 Patients in the Victorian Epidemic of Cerebro-Spinal Meningitis in 1915. (Fairley and Stewart.[9])

Age in Years.	Male.	Female.	Total.	Percentage.
Under 1	20	17	37	5·9
1 to 5 ..	46	37	83	13·2
6 to 10 ..	41	27	68	10·8
11 to 20 ..	118	34	152	24·2
21 to 30 ..	140	28	168	26·8
31 to 40 ..	33	9	42	6·7
41 to 50 ..	28	15	43	6·9
51 to 60 ..	15	13	28	4·5
61 to 70 ..	3	1	4	0·6
71 to 80 ..	1	1	2	0·3
Over 80	1	—	1	0·1
Totals ..	446	182	628	100·0

In Figure X the sex incidence in the various age groups in the Royal Prince Alfred Hospital series is shown. An inspection of this graph reveals a striking preponderance of males in the adolescent and early adult years. The only other point of great divergence is in the first quinquenniad, where males again predominate. In the figures given by Fairley and Stewart males predominate at all ages.

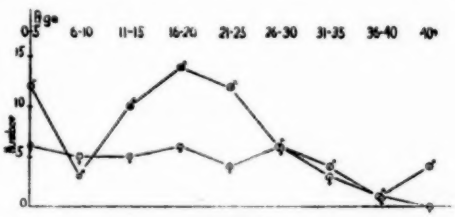

FIGURE X.

Graph showing the age and sex of patients suffering from cerebro-spinal meningitis admitted to the Royal Prince Alfred Hospital from 1910 to 1937. Note the preponderance of males over females in the years of adolescence and early adult life.

Geographical Distribution.

The distribution of cerebro-spinal meningitis is practically world-wide. The reason why some countries have suffered no major epidemics is that conditions for the rapid dissemination of infection have not been suitable when the infection has been introduced. It may be that there are some countries entirely free of infection. For example: during eight years in the Territory of New Guinea I never encountered a case of cerebro-spinal meningitis, and I have heard of no cases since I left that country.

PATH OF INFECTION IN THE BODY.

It has long been accepted that the causal organism gains entry to the body by way of the naso-pharyngeal mucosa. Various theories have been developed as to the pathway from the naso-pharynx to the meninges. Some have thought that the infection spread to the accessory sinuses, thence through the floor of the skull to the meninges. Others have suggested that there is a direct communication between the subarachnoid space and the lymphatic channels of the naso-pharynx. Others again have suggested that the infection travels along the sheaths of the branches of the olfactory nerves. It is now more or less generally accepted that the meninges are infected from the blood, that, indeed, there is invariably a stage of bacteriæmia or septicæmia. Some people have even gone so far as to declare that the organisms are carried to the meninges by phagocytes.

It is of interest at this point to recall the work of W. E. Le Gros Clark.[18] Clark found that fluids dropped into the nasal cavities of rabbits reached the surface of the brain within one hour, passing along the perineural sheaths of the olfactory nerves to the subarachnoid space. In his words: "The evidence indicates that the spaces of these perineural sheaths are continuous above with the subarachnoid spaces and extend peripherally along the peripheral fibres of the olfactory nerves to the olfactory sensory epithelium." He found no evidence that the subarachnoid space was in direct communication with the lymphatics of the nasal mucosa.

EPIDEMIOLOGY.

On the subject of epidemiology I have nothing to add to what has already been said under the headings of "History" and "War", except that we must be prepared for epidemics throughout Australia in July or August.

SYMPTOMS AND COURSE.

INCUBATION PERIOD.

The incubation period of cerebro-spinal meningitis is said to be usually three to five days,[19] but is apparently variable. Andrewes, Bulloch and Hewlett[10] state that it may be three or four days or as long as three weeks or more. They point out that when the naso-pharynx is invaded the immediate commencement of incubation is not inevitable. In a report issued by the British Ministry of Health in 1931,[20] it is stated that the incubation period is "indefinite, but usually from 3 to 5 days; . . . may be as long as 9 to 10 days and in some fulminating cases as short as 24 hours".

STAGES.

Most authorities[8][11][12][22] now recognize the existence of three stages in disease produced by the meningococcus: (i) catarrhal or naso-pharyngitic, (ii) bacteriæmic or septicæmic, (iii) meningitic or metastatic. This conception is consequent on the assumption that the organisms (i) invade the naso-pharyngeal mucosa and evoke an inflammatory reaction, and (ii) invade the blood stream, and (iii) are deposited in and multiply in susceptible tissues (for example, the meninges), where they cause suppuration.

The Catarrhal Stage.

Naso-pharyngitis is the first clinical evidence of invasion by the meningococcus; but it may not be observed in fulminant cases, in which invasion of

the blood and meninges may be so rapid that it has not time to develop before the patient is laid low. Some observers[10][22][23][24] regard naso-pharyngitis as incidental only. Bullock[24] remarked that as practically everyone had catarrh at some time during the epidemic period in England in 1915, not much importance could be attached to its concurrence with cerebro-spinal meningitis.

On the other hand, characteristic appearances of the naso-pharynx are described by E. G. D. Murray:[14]

> The acute stage of this pharyngitis may be very transitory and within two or three days may be difficult to detect. It is only in the fulminant case, as a rule, that the pharyngitis can be studied *post mortem*. The lack of evidence of constant pharyngitis in carriers examined by many authors may, therefore, bear some relation to the time elapsing between their infection and the date of the examination.
>
> Essentially the lesion is an acute congestion with an intense extravasation of leucocytes into the œdematous tissue. There is an acute catarrhal condition of the mucous membrane which is covered with tenacious muco-pus containing very numerous polymorphonuclear leucocytes. The whole picture is intensified over the pharyngeal tonsil, the crypts of which are plugged with muco-pus. The meningococci are abundant in the muco-pus and their numbers are greatest where the reaction is most intense.

In the great majority of cases individual resistance is so high that the invading forces are unable to penetrate beyond the naso-pharyngeal defences.

Fairley and Stewart[8] noted that pharyngitis was a predominant sign, but was usually symptomless.

Conjunctivitis is a not uncommon accompaniment of pharyngitis in the first stage.

The Stage of Bacteriæmia.

The second stage commences usually within forty-eight hours of infection; but it may be delayed for many days or weeks.[12] The onset is sudden, often marked by a rigor in adults and convulsions in children. In most cases the only symptoms are suggestive of nothing worse than influenza. During an epidemic of cerebro-spinal meningitis the medical man should be on his guard and should take measures to satisfy himself of the true significance of any influenza-like symptoms.

Lundie, Thomas, Fleming and Maclagan[21] give the following list of symptoms and signs:

> I. Headache (almost invariably present).
> II. Pains in back and limbs.
> III. Chilliness and nausea. (Vomiting occurs sometimes; in rare cases it is the only symptom.)
> IV. Mental depression or exaltation.
> V. Photophobia.
> VI. Orbital pain; pain on ocular movement.
> VII. Pyrexia (100° to 102° F.).
> VIII. Moderate acceleration of the pulse rate.
> IX. Intolerance of light and sound; hyperæsthesia; hydrocephalic cry; exaggerated reflexes; ankle clonus (frequently to be elicited); tetanic spasms when attempts are made to obtain some reflexes; acute mania (an occasional symptom).
> X. Rigidity of neck and head retraction (these are occasional symptoms).
> XI. Flushing of the skin; great sensitivity of the skin to touch and cold.
> XII. *Tâche cérébrale.*

Herrick[12] states that stupor may occur at this stage, and that petechial hæmorrhages into and around joints may occur, causing pain and intolerance of movement.

Some of these symptoms suggest that the meninges have already been invaded. It is impossible to draw any clear line of demarcation between the second and third stages. It is obvious that organisms will continue to circulate in the blood during the third stage.

The second stage ordinarily lasts about forty-eight hours, but may be very much shorter in fulminant cases. In some cases it lasts for several weeks.

The Stage of Meningitis.

The third or "metastatic" stage is the stage in which tissues remote from the point of entry are invaded by meningococci from the blood stream. In 90% of cases, says Herrick,[12] the dominant metastasis is meningeal; but the joints, pericardium, lungs, epididymis, skin or endocardium may be attacked. My concern is with the meningitic symptoms. Meningococcal lesions of some other tissues will be mentioned as complications only.

It must be borne in mind that some of the symptoms of the third stage are due to the blood infection only, having taken longer to develop than the manifestations of meningeal involvement. Also some of the symptoms of the second stage continue into the third.

A General View of the Clinical Manifestations.

The symptoms are mainly those of sepsis, toxæmia and raised intracranial pressure. There is always a history of headache and nearly always of vomiting. The temperature is raised and there may be a history of rigor or convulsions. The pyrexia is very irregular, tending to remissions and exacerbations. The patient may be either wildly delirious—perhaps maniacal—or stuporose or comatose. If he is stuporose he may be very irritable and resentful of being handled. The "meningeal" or "hydrocephalic" cry may be in evidence. Stiffness of the muscles of the back of the neck and the spine is present. Head retraction is the rule. Kernig's sign and Brudzinski's signs can be elicited. The pulse rate is variable, but tends to be slower than the temperature would lead one to expect. There may be strabismus or inequality of the pupils. Labial herpes is frequently present. A petechial rash occurs in perhaps 40% of cases. The "elbow sign" described by Fairley and Stewart may be present. Other types of cutaneous eruption may occur (see below).

Lumbar puncture reveals turbidity of the cerebro-spinal fluid. Gram-negative intracellular diplococci are found on microscopic examination.

According to Fairley and Stewart,[8] the blood pressure is apt to be low in the early stages of the illness, when the patient is overwhelmed by toxæmia, but rises later as the intracranial pressure rises. There is a polymorphonuclear leucocytosis.

Description of Important Signs.

Head Retraction and Neck Stiffness: According to Fairley and Stewart, Cecil Wall[25] states that in flexion and extension of the head the cerebellum slides up and down the posterior surface of the medulla and unfolds and folds that part of the arachnoid that forms the bridge between them. If the arachnoid is inflamed, the muscles at the back of the neck become fixed in contraction to prevent this movement. In the Victorian epidemic of 1915 and 1916 stiffness of the neck was the most frequent of all signs of meningitis. The neck and back may be so rigid that any vigorous attempt to flex the neck results in lifting of the whole trunk. Retraction of the head (dorsiflexion beyond the vertical) was not common in the Victorian epidemic.

Kernig's and Brudzinski's Signs: Kernig's sign is elicited by flexing the hip to a right angle and extending the knee. If the sign is present the knee cannot be extended much past a right angle. Next to stiffness of the neck this is perhaps the most helpful sign in diagnosis. When one lower limb is passively flexed at the hip and knee the other may be flexed involuntarily. This is Brudzinski's leg sign. To elicit Brudzinski's neck sign, the medical attendant attempts to flex the head while holding the chest; the lower limbs are involuntarily flexed at the hips and knees.

Rashes: The petechial or purpuric rash is the one usually described. It may occur before the onset of meningitic symptoms. It appears most profusely on the trunk. The face is affected only when the infection is very severe. Petechiæ or purpuric patches may coalesce. Blisters, at first containing clear fluid, but later blood, may appear over the larger patches. Macular erythematous blotches are occasionally seen. In some cases petechiæ appear in the centres of the macules.

Fairley and Stewart describe what they term the "elbow sign", which appears on the skin covering the elbows, the great trochanters, the medial aspects of the knees, and the shoulders.

> The actual lesion consists in a pronounced flush on the extensor aspect of the joint, which at first appearance might be mistaken for a pressure mark, but is differentiated therefrom by the fact that, especially in the case of the elbows, it covers a wider area than would be actually subjected to pressure.

Sometimes the "elbow sign" is associated with what Fairley and Stewart call "hæmorrhagic gooseskin" of the same areas.

An herpetiform eruption in the circumoral region is common.

The Mental State: In a few cases the mental state remains practically normal throughout; but in the majority there is a profound disturbance for a longer or shorter period. In the early stages of the illness there may be excitability and hyperactivity; later the manifestations of a disordered mind may vary from a mild disorientation to wild mania. Stupor or coma may follow delirium or appear early in the illness in the absence of delirium. A feature of a patient in a stupor is his extreme irritability and resentment of being touched. Douglas[7] remarks on the change from delirium and headache at the onset to a more optimistic state of mind, perhaps after the first lumbar puncture.

The Cranial Nerves: Twitching or spasm of the facial muscles is a frequent symptom. Strabismus is not uncommon. Examination of the *fundus oculi* may reveal papillœdema. Any of the other cranial nerves may suffer.

Temperature: The temperature is irregular (see Figure XI). The severity of the illness cannot be measured by the height of the pyrexia; but hyperpyrexia is apt to occur immediately before death (see Figure XII). Occasionally the temperature chart resembles that of benign tertian malaria. Rigors and sudden pyrexial paroxysms may occur during the course of the illness.

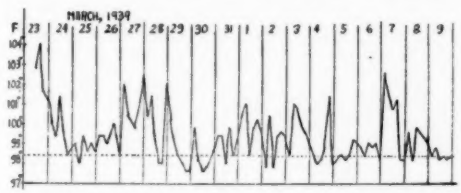

FIGURE XI.

Temperature chart of a patient (see Case I) suffering from cerebro-spinal meningitis before the introduction of the sulphanilamides. (Obtained from the Sydney Hospital records.)

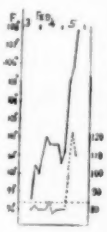

FIGURE XII.

Temperature chart showing hyperpyrexia immediately preceding death. (Obtained from the Sydney Hospital records.)

Pulse: In the early stages the pulse may be rapid and soft. Later it becomes fuller and slower.

Cerebro-Spinal Fluid: The cerebro-spinal fluid obtained at lumbar puncture is under increased pressure and is usually turbid with pus cells. Microscopic examination or culture reveals the meningococcus.

TYPES OF THE DISEASE.

The types are fulminant or malignant, acute or ordinary, abortive, and chronic.

The Fulminant Type.

It is said that the fulminant type of the disease occurs in a higher proportion of cases in the early stages of an epidemic; it also occurs sporadically. The onset is sudden. Mental disturbance occurs early and is profound. A purpuric rash usually appears. The temperature may be subnormal or moderately raised in the early stages; later it may soar to a great height or fall to subnormal. Formerly these cases all terminated in death within forty-eight hours. Osler[26] states that "Stillé tells of a child of five years, in whom death occurred after an illness of ten hours, and refers to a case reported by Gordon, in which the entire duration of the illness was only five hours".

The Acute Type.

The acute type of the disease follows a very variable course. Its duration might be several days

or several months. In the more prolonged cases remissions and exacerbations are likely. Whether the illness is short or long, convalescence is apt to be tedious. Most of the deaths occur within the first five days.

So one would have written before the introduction of the sulphanilamide group of drugs. Now the story is very different, most cases terminating rapidly.

The Chronic Type.

Favourable as all the reports of the therapeutic value of sulphanilamide preparations have been, it seems almost too much to hope that there will be no chronic cases. This type of the disease persists for months, the patient becoming more and more wasted, subject to a variable degree of pyrexia and remissions and exacerbations. The death rate is high.

The Abortive Type.

The abortive type may set in suddenly, with high fever and acute symptoms, but terminate within twenty-four or forty-eight hours, without treatment, or it may be mild and evanescent and unrecognizable save in an epidemic. It is of interest to note here that meningismus has been observed in carriers. This may have been due to a mild attack of meningitis, or it may have been an hysterical manifestation similar to the symptoms observed in some unstable and frightened persons in an epidemic of poliomyelitis.

DIAGNOSIS.

Little more need be said here on diagnosis. It will in the end depend on the appearances of the cerebro-spinal fluid. But if the symptoms are clearly those of meningitis and no abnormality of the cerebro-spinal fluid is found at the first examination, the diagnosis of meningitis must not be immediately discarded. It sometimes happens that the fluid is clear at the first tapping.

In the differential diagnosis the following may give rise to some difficulty: meningitis due to organisms other than the meningococcus, poliomyelitis, heat-stroke (in places where this occurs), local inflammatory conditions causing stiffness of the neck, rigidity of the neck due to spinal disease, other infectious diseases, such as influenza and typhoid and pneumonia, uræmia, and intracranial lesions. If joints are swollen and painful a wrong diagnosis of rheumatic fever is possible.

As a rule diagnosis will not be very difficult. The danger is to neglect the possibility that the illness may be cerebro-spinal meningitis.

COMPLICATIONS.

Complications are numerous.

Bed-Sores: Rapid emaciation and the prominence of bony protuberances, aided by trophic changes and incontinence of urine and fæces, are apt to lead to the formation of bed-sores in the absence of efficient nursing.

Vomiting: Vomiting is a symptom rather than a complication. If it persists it should be regarded as evidence of hydrocephalus.

Hæmatemesis and Melena: Hæmatemesis and melena are not frequently seen. They are manifestations of the tendency to bleeding, as evidenced also by the purpuric eruption.

Cardiac Lesions: Pericarditis caused by meningococci is an occasional complication. Vegetative endocarditis may occur.

Lymphadenitis: Fairley and Stewart[8] noted cervical lymphadenitis in nearly 50% of convalescents during the Victorian epidemic of 1916, but none in 1915. "It takes the form of a more or less brawny swelling of the glands of the neck. the salivary glands participating; there does not appear to be any tendency to pus formation."

Pulmonary Complications: Bronchitis, pleurisy, bronchopneumonia and lobar pneumonia are not infrequent complications.

Infection of the Urinary Tract: Pyelitis and cystitis are common. Pyuria, with or without symptoms, is frequently seen.

Arthritis: In some epidemics arthritis has been a frequent complication. As a rule several joints are affected at once. Pus may form in the joints. In some cases, as mentioned earlier, swelling of joints may be due to extravasation of blood.

SEQUELÆ.

The number and type of sequelæ vary in different epidemics. In the Victorian epidemics of 1915 and 1916 it was found that as a rule recovery was complete.

Hydrocephalus is perhaps the most important of the sequelæ. It is most likely to occur in subacute and chronic cases. It should be rare now that the sulphanilamide drugs are employed freely in treatment. It is usually fatal. In some rare cases the normal circulation of the cerebro-spinal fluid may be restored and recovery may ensue. Mental deterioration, fits and persistent headache may follow the illness. It may be that they are due, in some cases at any rate, to hydrocephalus. Deafness may occur, either as a sequel to *otitis media* or as nerve deafness. Hemiplegia and various cranial nerve palsies may occur during the fever, and in some cases persist. Blindness may follow papillœdema.

PROGNOSIS.

The prognosis has altered completely since the introduction of the sulphanilamide group of drugs. In the older epidemics the death rate was from 20% to 75%. From 1910 to 1937, 97 patients were treated in the Royal Prince Alfred Hospital; 43 died (see Figure XIII). In most of these cases, even as far back as 1910, antiserum was used—perhaps not always as vigorously as it might have been. The best results from serum therapy that I have been able to find recorded were reported by Herrick.[27] Of 137 patients treated by him by the intrathecal administration of antiserum, 47 (34·3%) died; of 128 treated by intrathecal and intravenous methods, 19 (14·8%) died. But he recognized the infection in the stage of bacteriæmia in 105 of the 265 cases. It may be that many of these would not have gone on to meningitis.

Now the mortality rate is amazingly lower. Banks[28] treated 65 patients with antiserum and sulphanilamide (one with sulphapyridine), eight dying; of a further six who were inadequately treated, four died. In a later series he treated 31 with sulphanilamide alone, 36 with sulphapyridine alone, and five with both sulphanilamide and sulphapyridine, only one patient dying; a further four were inadequately treated and all died.

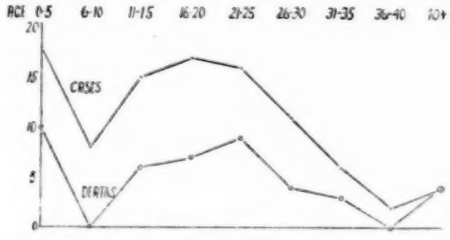

FIGURE XIII.

Graph showing the death rate in various age groups of persons treated in the Royal Prince Alfred Hospital for cerebro-spinal meningitis during the years 1910 to 1937.

The most remarkable results were obtained by Somers[29] and Bryant and Fairman,[30] in Africa. Somers states that from 1934 to 1938 there were 21,599 cases and 14,816 deaths in the Anglo-Egyptian Sudan. He treated 143 patients with remarkably small doses of sulphapyridine administered intramuscularly, intrathecally or intraperitoneally, and only 14 of them died, though some were apparently moribund. Bryant and Fairman reported nine deaths in 189 cases among natives of the Sudan.

The mortality rate is said to be higher among infants under twelve months of age and adults over 35 years than among people of intermediate ages. From 1914 to 1939, 230 children suffering from cerebro-spinal meningitis were treated at the Royal Alexandra Hospital for Children, Sydney; 136 of them died. There was thus a mortality rate of 59·1%. In 1938 and 1939, when sulphanilamide compounds were used, only two of thirteen died.

PROPHYLAXIS.

General.

Overcrowding.

Enough has been said under the heading of "Ætiology" to stress the importance of avoiding overcrowding as the first essential in prophylaxis. The experience of the Great War of 1914-1918 should be sufficient to make overcrowding in military encampments and institutions, except in grave emergencies, almost a criminal offence.

Ventilation.

In cold weather ventilation is often inadequate. Even men sleeping in tents may have a poor supply of oxygen, as they are inclined to close up every crevice that the thinnest stream of air could trickle through. Open windows of barracks, huts and dormitories, and open doors of tents should be insisted on. In Australia in all but really atrocious weather, tent flies can remain rolled. The value of fresh air and sunshine in diminishing the carrier rate has been amply proved.[31]

Lowered Resistance.

It has been suggested that the recruit should be allowed to settle down in his new conditions of army life before he is subjected to vaccination and inoculation, and that he should not be called on to carry out strenuous physical exercises during the early days after his enlistment. It might be added that men suffering from the effects of vaccination or inoculation or from any infection, such as a cold, that would tend to lower the resistance, should be put off duty. Nurses and others attending to persons suffering from cerebro-spinal meningitis should be similarly protected.

Isolation of Patients.

The incidence of cerebro-spinal meningitis is low; therefore isolation of patients is relatively easy. Isolation is an obvious precaution, as it is one means of preventing the access of more than a limited number of susceptible people to a known source of virulent organisms. On the other hand, it does little to limit an epidemic; for, after all, meningitis is merely the occasional clinical manifestation of a widespread epidemic of naso-pharyngeal infection.

Carriers and Contacts.

Segregation.

Except in institutions, the segregation of carriers is impracticable: there are too many of them. The segregation of contacts during an epidemic seems to be scarcely worth the trouble. If they are carriers they are no more likely to transmit the infection than any other carriers. The only argument in favour of the segregation of contacts is that they are perhaps more likely than other carriers to harbour virulent organisms.

Disinfection of the Naso-Pharynx by Antiseptics.

During the Great War spraying of carriers' throats with various antiseptic solutions was employed. Several methods of mass treatment were evolved. A small room was saturated with antiseptic vapour from a steam atomizer, and carriers breathed the atmosphere for five to ten minutes.[32] Gordon found that the treatment of chronic carriers by this means was not satisfactory, but that it increased the rate of elimination of temporary carriers.

Treatment of Carriers with Drugs of the Sulphanilamide Group.

The treatment of patients with drugs of the sulphanilamide group has had such happy results that medical men have been encouraged to administer the same drugs to carriers. This method is hardly suitable where large numbers are being dealt with, but may be of great value in an institution. Meehan and Merrilees,[33] who have allowed me to quote their results, employed sulphapyridine in the treatment of carriers in a foundling hospital, where epidemics had recurred yearly for several years. They gave a dose of one gramme per 14

pounds of body weight per day, for five days; they then allowed five days' rest, then gave a total of ten grammes per 14 pounds of body weight during a fortnight. Of 75 children treated in this manner, six still harboured the organism after one course, four after two courses, and three after three courses After the lapse of a year there has been no recurrence of the disease.

Active Immunization.

Banks[34] has discussed the possibility of producing active immunity by repeated injections of Ferry's exotoxin.[35][36][37][38] The intradermal injection of this toxin has also been used, like the Dick test and Schick test, to reveal susceptibility. There are interesting possibilities here. As far as I know, insufficient work has been done to prove the value of either of these procedures.

Vaccines have been used from time to time; they are of doubtful value.

Personal Prophylaxis.

Medical attendants, nurses and orderlies, engaged in the treatment of persons suffering from cerebro-spinal meningitis, should have adequate hours of rest and should have quarters that are well ventilated; they should take daily exercise in the open air and sunshine. It has been asserted that they should wear masks. But, while such a procedure would probably ensure protection against the organisms, it might have a bad moral effect on the patients. Whether sulphapyridine should be taken by medical and nursing attendants as a prophylactic is difficult to decide at present. I should say that it should not be taken except by persons who are found to be carriers. The recognition of the carrier state in the medical attendant appears to be of special importance because of the chances of his transmitting the infection to people whose resistance is already lowered by some other illness.

TREATMENT.

General.

There is no need for me to stress the importance of efficient nursing. All sources of irritation should be removed. Special care should be taken of the bony prominences. If the patient is incontinent, cleanliness is of particular importance. If possible the use of mechanical restriction should be avoided. Such a thing as a restriction sheet must be a source of great annoyance to a patient already in an irritable state. Morphine is probably the best for vomiting, pains and restlessness.

Lumbar Puncture and Cisternal Puncture.

Lumbar puncture should always be performed, as a therapeutic measure as well as for diagnosis. Formerly it was repeated daily or oftener; but since the introduction of the sulphanilamide group of drugs many repetitions have become unnecessary. Lumbar puncture should be performed whenever there is evidence of meningeal irritation. From 30 to 75 or even 100 cubic centimetres of cerebro-spinal fluid may be run off at one puncture. Frontal headache commonly occurs even when a small amount of fluid has escaped; it continues until some serum or other fluid has been run in.

In those rare cases in which fluid cannot be obtained by lumbar puncture, cisternal puncture should be done.

Antiserum and Antitoxin.

The introduction of intensive treatment with antiserum was responsible for a great decrease in the mortality rate. Antiserum had been in use for some years before the Great War; but it was not until the great epidemics of 1915 and 1916 that its real value was demonstrated; for it was not until then that it was generally used in sufficiently large doses. It is given intravenously in the bacteriæmic stage and intrathecally or both intrathecally and intravenously in the stage of meningitis. Herrick[27] advises the administration of large doses (50 to 150 cubic centimetres) intravenously and smaller doses intrathecally. He states that much less serum need be administered intrathecally if large intravenous doses are given. The amount of antiserum run into the theca should not exceed the amount of cerebro-spinal fluid run off. Some workers irrigate the theca with saline solution before injecting antiserum. In this procedure the patient is tipped up and back, to allow the irrigating fluid to flow along the cord and back by gravity.

Hoyne[39] obtained better results by treatment with Ferry's meningococcal antitoxin than with antiserum.

Chemotherapy.

The sulphanilamide drugs have revolutionized the treatment of cerebro-spinal meningitis. Some of the results have been so good as to be almost unbelievable. Now it seems that the only persons likely to die of cerebro-spinal meningitis are: (i) those who have some other disease, (ii) those who are overwhelmed early in the fulminant type of the disease, and (iii) those to whom specific therapy is given too late.

The drug that is most freely used in British communities is sulphapyridine (2-sulphanilylamidopyridine, "M & B 693"). Whitby,[40] as a result of his experiments on mice, concluded that sulphanilamide was effective against meningococcal infections, but that "Proseptasine" and "Soluseptasine" were not. On the other hand, Hannah and Hobson[41] obtained good results with these two drugs in a small series of cases. Banks[42] states that "Prontosil" and "Prontosil Soluble" are comparatively slow to reach the cerebro-spinal fluid. At first, both antiserum and sulphanilamide were given. Now the tendency is to administer sulphapyridine alone. There seems to be nothing against the administration of antiserum.

Banks[28] states that sulphanilamide should be given in a sufficiently high dosage to produce a concentration of 5·0 milligrammes per 100 cubic entimetres of cerebro-spinal fluid; he suggests that such a high concentration may not be necessary when sulphapyridine is used. Hobson and

MacQuaide[43] state that a concentration of 3·0 milligrammes per 100 cubic centimetres of sulphapyridine appears to be adequate. They believe that the permeation of the chorioid when it is inflamed is achieved more readily than when it is intact.

In a pamphlet[44] issued recently by the War Office the importance of early treatment is stressed. The institution of specific therapy must not be delayed until lumbar puncture has been performed. The first dose should be 1·0 gramme of sulphapyridine given intramuscularly. If the patient is unconscious this should be repeated every four hours. If the patient is conscious he is given 1·0 gramme by mouth two hours after the injection, then 2·0 grammes every four hours until about nine grammes or a total of 1·0 gramme per stone of body weight have been given. This suffices for the first period of twenty-four hours. A similar amount should be given daily for several days, depending on the response.

In fulminant cases 1·0 gramme (three cubic centimetres) of "M & B 693 Soluble", diluted in three or more volumes of normal saline solution, should be given intravenously and 1·0 gramme intramuscularly immediately; thereafter 1·0 gramme should be given intramuscularly every four hours. Ample fluids should be administered.

An intramuscular injection of this drug causes pain; therefore local anæsthesia is desirable.

Children require relatively larger doses than adults. The following scheme of dosage is given by Banks.[28]

Age in Years.	Dose in Grammes Per Day.
0	3·0
2	4·5
5	6·0
10	7·5
15 and over	9·0

Banks[42] also states that infants should be given 3·0 grammes and children 1·5 grammes per 14 pounds of body weight a day.

Somers[29] and Bryant and Fairman,[30] in Africa, gave remarkably small doses with almost incredible success.

Other drugs scarcely need mention. If there is anyone who still imagines that hexamine is of any use in the treatment of infection of the meninges, I would refer him to the work of Fairley and Stewart.[8] Hexamine depends for its effect on the liberation of formalin in an acid medium. Fairley and Stewart showed that however much acid was given by mouth the cerebro-spinal fluid remained alkaline.

Records of Cases.

The following two reports of cases were obtained from the records of Sydney Hospital.

CASE I.—A boy, aged eleven and a half years, had been ill for four days prior to his admission to Sydney Hospital on March 23, 1929. The illness commenced suddenly, with headache and vomiting, which lasted for half a day. Two days later he went to school. He suffered a recurrence of vomiting and headache that night. At the time of his admission to hospital he was restless and flushed. He was unable to recognize his parents. Kernig's and Brudzinski's two signs were present. At lumbar puncture 40 cubic centimetres of turbid cerebro-spinal fluid were withdrawn. The fever continued irregularly until April 10, when it subsided. The cerebro-spinal fluid was still turbid on April 7. Antiserum was given daily to that date (see Figure XI).

CASE II.—On the night of August 26, 1924, a girl, aged fourteen and a half years, felt ill and fainted. Her face twitched before she regained consciousness. At 2 a.m. on August 27 she commenced to scream, calling out for relief from pain in her head. She continued to scream until the time of her admission to Sydney Hospital at 6 p.m. that day. According to the hospital record she had a meningeal cry; her left eyelid was drooping and she had a slight left internal strabismus. Purulent fluid under increased pressure was obtained at lumbar puncture. Meningococcal antiserum was given intrathecally under chloroform anæsthesia. She died on August 31, 1924 (see Figure XIV).

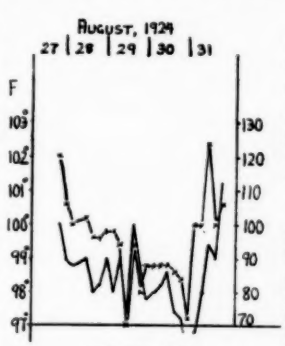

FIGURE XIV.
The temperature (continuous line) and pulse chart of a fatal case of cerebro-spinal meningitis. Obtained from the Sydney Hospital records.)

How different is the story now that the value of the sulphanilamide drugs has come to be recognized. The following report was obtained from the records of the Prince Henry Hospital.

CASE III.—A girl, aged nineteen years, was admitted to the Prince Henry Hospital, under the care of Dr. R. Jeremy, on December 31, 1939. She had suffered from headache for the previous twenty-four hours and had been restless for six hours. She appeared to understand what was said to her, but gave no answer to questions. She was unable to recognize people. Her reflexes were normal. She had no rigidity. Kernig's sign could not be elicited. She was given four intramuscular injections, of 1·0 gramme each, of "M & B 693".

The next day (January 1, 1940) the patient still had severe headache and was stuporose; weakness of the right facial muscles was noted; the right knee jerk was weaker than the left; rigidity of the neck was pronounced. At lumbar puncture at 11 a.m. the cerebro-spinal fluid was found to be turbid and under increased pressure; a quantity was drained off. At 8 p.m. the pressure of cerebro-spinal fluid was still found to be above normal. Bacteriological examination of the cerebro-spinal fluid revealed the presence of meningococci. On this day 7·0 grammes of "M & B 693" were given by mouth.

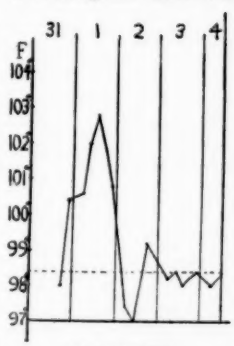

FIGURE XV.
Temperature chart of Case III.

On January 2 right external strabismus was noted. Kernig's sign was elicited on both sides. There was a concentration of 10·0 milligrammes per 100 cubic centimetres of sulphapyridine in the cerebro-spinal fluid. The temperature was 97·4° F. in the morning and rose to 99·2° F. in the afternoon.

On January 3 "M & B 693" was given in a dose of 0·5 gramme every four hours. There is no record of its further administration. Convalescence was uneventful. The patient was discharged from hospital on January 23, 1940 (see Figure XV).

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Reviews.

MINOR SURGERY.

IN the preface to his book "Minor Surgery" R. J. McNeill Love is conscious of the difficulties regarding the scope of the word "minor".[1] His handling of the material is admirable. But he presents very little subject matter that cannot be found in any surgical manual.

The main virtue of this interesting little book is the presentation of necessary knowledge in a concise, easily readable form. It commences with a general discussion on the examination of the patient, the principles of asepsis and antisepsis, and preoperative procedures, and then proceeds to discuss wounds and burns. This is followed by a chapter on the vascular system, including the treatment of hæmorrhage and a concise account of bandaging, knots and strapping.

The remainder of the book deals with minor operative procedures, such as blood transfusion, the administration of saline solution by the drip method, *paracentesis abdominis, et cetera*, and minor operations on the ear and eye, and the emergency treatment of the common fractures. There are also chapters dealing with hernia and the genito-urinary system and, in a general way, anæsthesia.

While this latest production by Dr. McNeill Love will be of considerable interest to students and resident medical officers, we feel that it presents no advantages over recognized text-books and falls below the standard set by the author in his "Short Practice of Surgery".

[1] "Minor Surgery", by R. J. McNeill Love, M.S., F.R.C.S.; 1940. London: H. K. Lewis and Company Limited. Crown 8vo. pp. 378, with 155 illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, JULY 20, 1940.

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AUSTRALIAN MEDICAL PRACTITIONERS AND THE WAR.

ABOUT two years ago the Defence Department issued to medical practitioners in Australia a *questionnaire* asking them to state their qualifications and to indicate whether they would be available for service. At a recent meeting of the Victorian Branch of the British Medical Association Major-General R. M. Downes, Director-General of Medical Services, said that 6,450 copies of the *questionnaire* had been addressed to medical men and women and that 5,800 replies had been received; 80% of those who replied were available for service of some kind. In other words, there are in Australia 4,640 medical practitioners who are able and have expressed their willingness to serve the State in this critical time. Major-General Downes also told his audience something about the Committee for the Coordination of Medical Services which had been set up in 1938. On this committee are representatives of the medical services of the Navy, Army and Air Force, of the administrative staff of the Army, of the Commonwealth Department of Health, of the British Medical Association and of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons. In each State there is a branch or subcommittee of the central committee under the chairmanship of the Deputy Director of Medical Services, consisting of representatives of interested bodies and having powers of cooption. In certain of the States regular meetings of the State coordination committees have been held and useful work has been done. Most of the activities of these committees are of necessity in large measure confidential, but Major-General Downes has stated that they are concerned with personnel and with civil requirements on mobilization. Satisfactory though these arrangements appear to be, it must be remembered that the information contained in the *questionnaires* sent out by the Defence Department is two years old, and that in the meantime many changes have taken place. This has been the occasion of much thought by certain sections of the medical profession, who have realized that the time may shortly come when the authorities may find it useful to have at hand detailed, reliable and up-to-date information about the circumstances and attainments of every medical practitioner. The medical profession was best qualified to collect information about its own members, so it was believed, and the coordination committees or the authorities themselves would welcome detailed and collated facts, should they suddenly be required. The Federal Council of the British Medical Association in Australia has therefore determined to send out to medical practitioners in all the States a second *questionnaire,* asking for its return as soon as possible. The information sought is more detailed than that obtained in the first *questionnaire,* and, correctly supplied, would go a long way towards enabling a controlling body to reorganize medical practice in such a way as to satisfy the civil and military needs of the community if such a step should be necessary.

Medical practitioners must clearly understand the nature of the present inquiry. It is an inquiry by the medical profession for information from its own members, and is made in order that the profession may be able, should it be necessary, to supply the authorities with all essential facts. In

this sense it is a private inquiry by a body anxious to see that its resources are used to the best possible advantage. There are several other points to be emphasized. In no possible way can the inquiry be regarded as an encroachment on the departmental sphere of activity. Moreover, it is not an appeal for service, either at home or abroad, since large numbers of practitioners have already offered for both spheres. This statement is necessary on account of the remarks reported recently in the public Press as having been made by the Honourable G. A. Street, Minister for the Army, when he stated that women were to be given commissions in the Australian Army Medical Corps because of the shortage of men—a reflection on the men not justified by the facts. One effect that we hope this inquiry will have is to set at ease the minds of many practitioners who want to do something and who are impatient because their offers have not been promptly accepted. The knowledge that their qualifications and their claims to be allowed to undertake active work are to be set out in detail for eventual consideration by those in authority may make these practitioners content. Medical practitioners may be happy in the knowledge that their own association is doing its utmost to ensure that Australian medicine shall be ready to meet all possible eventualities.

Current Comment.

THE MOLECULAR THEORY OF ORGANISMS.

SUCH "special articles" as that which *The Lancet* has recently published on viruses,[1] setting out briefly the latest additions to our knowledge of the nature and properties of these bodies, must be widely appreciated by the medical profession. Discoveries of far-reaching importance, which must be stimulating to workers in many fields of medical science, tend to escape our notice for a long time when they are made by pure biologists and plant pathologists, who have a scientific literature of their own.

In the past there has been a good deal of speculation as to whether viruses are living things or not—that is to say, as to whether they are some strange form of organism or whether they are of the nature of enzymes, toxins or products of cellular metabolism. It would appear from recent publications that all substances possessing virus activity which have so far been isolated have proved to be homogeneous, protein in nature and of very high molecular weight (of the order of at least 400,000). In the last few years variant forms of many viruses have been found in Nature and produced artificially. G. M. Findlay, writing in 1939, stated that he could find no essential difference and certainly no clear dividing line between the types of variation encountered in multicellular and unicellular organisms on the one hand and animal and plant viruses on the other, despite the fact that neither in bacteria nor in viruses is there clear evidence that hereditary characteristics are transmitted by structures of the type of chromosomes or genes. He concluded that although variation may not be an exclusive property of living organisms or even of organic compounds, the close analogy between the variations found in animal viruses and those in animals and plants makes the adoption of a vitalistic conception of the nature of animal viruses almost inevitable. The writer of the article in *The Lancet*, summing up, remarks that the question of whether viruses are living or not is of little importance, for "the word 'life' is merely a definition of degree", but that nevertheless study of the nature and mode of action of viruses may yield knowledge on the problems of normal and abnormal metabolism and on the nature of life. The ability of organisms to reproduce is associated with nuclear material and it is assumed that the basic unit of this material is a gene, perhaps a single nucleoprotein molecule: all viruses appear to be, or at least to contain, nucleoproteins. From this W. M. Stanley has recently concluded that the properties of viruses composed of nucleoproteins are essentially the properties of a gene or a group of genes, were these capable of a separate existence. He suggests that the characteristic structure which permits a virus to enter into the metabolic chain of events within cells may have a distinct bearing on the advent of cancerous growths. Stanley believes that there is really a continuum from simple to complex structures, from molecules to organisms, and that after all there is no great difference between the two.

This conception recalls very vividly a theory propounded some eight years ago by T. L. Cleave in a pamphlet which received little attention at the time but which repays study because of its prescience and infectious freshness of thought.[1] Cleave has taken up his stand one step ahead of Stanley; his theory is that any organism, however complex, is a single chemical molecule. This theory is based on the observation that there is frequently and the suggestion that there is universally a protoplasmic continuity between the cells of multicellular organisms. In the developing ovum, for instance, fine anastomosing threads of hyaline protoplasm may be demonstrated connecting the apparently separate cells of the morula; and it is usually

[1] *The Lancet*, March 30, 1940.

[1] T. L. Cleave: "Theory that an Organism is a Single Chemical Molecule", 1932.

accepted that the divisions of the nuclei in cell cleavage are not accompanied by complete division of the surrounding protoplasm and that the cell mass resulting from cleavage is a continuous whole. Actually this continuity might be postulated, for without it it is hard to see how the cells could divide so that the resulting formations always assumed the same positions relatively to one another. Again, the various syncytia of the body, the fine communications between the cells in the matrix of cartilage, the cell bridges between the prickle cells of the skin, the basement membranes of epithelia, all provide examples of demonstrable protoplasmic continuity in multicellular structures. Cleave's theory rests secondarily on the presence of a single consciousness in multicellular organisms. On the basis of his theory Cleave explains very simply such physiological phenomena as animal heat, movement, pleasure, pain and mind, pointing out that in their relation to the individual all these bear close resemblances to molecular reactions in chemical and physico-chemical processes. For instance, he draws an analogy between an organism developing a neoplasm and a molecule becoming chemically unstable at one point and breaking down so that a second smaller and different molecule results there. This second molecule may grow by stable polymerization, a process similar to growth in a simple multicellular organism, which is analogous to the growth of a benign neoplasm; or it may grow by unstable polymerization, a process similar to reproduction in a unicellular organism, which is analogous to the cellular proliferation in a malignant or potentially malignant neoplasm.

Such theorizing is worthy of attention, if it only tickles our fancy and frees our minds from the shackles of current conventional thought—more so if it leads us to grope further at important truths still hidden from us. Wherever this molecular theory of organisms may lead, perhaps we see in it the rationalization of the instinctive practice of every good and successful doctor to look beyond the physical ailment or the afflicted spot and minister to his patient as a human being.

CARDIAC PAIN.

PAIN may be exaggerated or produced by anxiety and diminished or completely subdued by the anxiety's removal. Cardiac pain is no exception. Geoffrey Bourne and E. Wittkower have conducted an experiment in the psychological treatment of patients suffering from angina of effort and patients suffering from "functional cardiac pain".[1] In a previous paper these workers described *angina innocens* as pain of cardiac origin and not due to intercostal fibrositis. In the paper now under review "the less controversial terms" "functional pain" and "functional cardiac pain" are used. They treated by psychological methods seven patients suffering from functional cardiac pain and five suffering from angina of effort. The distinction between the types is shown by a comparison of the two histories below. The first refers to a patient with functional pain:

> A woman of twenty-two had for the previous five years felt pain on exertion on the left side of the chest radiating to the neck and arm. It was increased after exertion but not during it, and was accentuated by worry and fatigue. There was no cardiovascular abnormality except slight accentuation of the first sound.

The second refers to a patient with angina of effort:

> A man of sixty-five had suffered for two years from a retrosternal pain that only came on as he walked and was proportional in its severity to the amount of walking. It was not increased by emotion, but was always accentuated by hills. On examination there was no increase in the blood pressure, but the apex beat was slightly displaced to the left, the heart/chest ratio being 14/27 cm. The electrocardiogram showed left-sided preponderance and some notching in the *R* wave in lead II.

The psychical disorders noted were hysteria, depression, anxiety and disorders of character. Treatment was aimed at helping "the patient to bring out and to face attitudes, previously unrecognized, which are often strongly emotional". Each patient was treated for one hour on three days a week. In all but a few cases the period of treatment was limited to 60 sessions, equivalent to 20 weeks. This seems a long time; but it is not unduly long, if it is effective, when compared with the years that may be occupied with fruitless physical treatment. The twelve patients investigated by Bourne and Wittkower had been treated by ordinary means for periods varying from one to thirteen years. Six out of seven patients were cured of functional pain. The remaining one experienced no diminution of pain, and she was the only one in the series who did not improve psychologically. However, she improved after the cessation of treatment and was able to return to work. One patient, suffering from hyperpiesia, died eight months after treatment. Two of the five patients suffering from angina of effort lost their pain, but suffered a relapse in six months; two experienced great relief, which continued, and the remaining one was unrelieved. Bourne and Wittkower note that psychotherapy has little or no effect on organic cardio-vascular disease, and they remark that removal of the pain—the essential warning against over-exertion—might be regarded as dangerous; but none of their patients with angina of effort died during a long follow-up period, "and, in fact, as the anginal attacks may in part be due to spasm of the coronary arteries, removal of these attacks may result in an improved circulation in the coronary system". At first thought it seems absurd that pain of organic origin can be completely removed by psychological means; but further consideration reminds us of the importance of anxiety in the causation of vascular disease, and we are no longer derisive. Psychotherapy is worth while in certain cases. It is of interest to conjecture on its possible effects on the state of the coronary vessels.

[1] *The British Heart Journal*, January, 1940.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Dysmenorrhœa.

O'DONEL BROWNE (*The Journal of Obstetrics and Gynæcology of the British Empire*, December, 1939) divides dysmenorrhœa into ovarian, uterine and mixed types. In the last mentioned the pain experienced is due to the presence of both types of painful menstruation. Ovarian dysmenorrhœa is characterized by a continuous severe premenstrual pain in the lower part of the abdomen or back, and exists by itself in about 10% of all cases. It occurs for two to three days before the flow begins and rarely persists after it commences. To differentiate between the two types the author relies on the history, on bimanual examination of the ovaries and the passage of a sound into the uterus. By comparison of the pain caused from palpation of the ovaries and that caused by the passage of a sound with the patient's account of her own pain it is possible to decide whether the dysmenorrhœa is of ovarian or uterine origin or whether it is a combination of both types. The treatment of this ovarian type is bilateral division of both infundibulopelvic folds at the brim of the pelvis, the nerves and blood vessels being severed in that situation. This is done under a general anæsthetic with the use of a mid-line incision. The underlying pathological change is stated to be sclero-cystic ovarian disease. If uterine dysmenorrhœa is also present, the treatment recommended is Cotte's presacral neurectomy. Cotte's operation in the hands of its author will produce only a 60% cure rate in severe painful menstruation, and the author submits his hypothesis of ovarian dysmenorrhœa as one of the reasons for this limited application of Cotte's method.

A New Classification of Ovarian Tumours.

WALTER SCHILLER (*Surgery, Gynecology and Obstetrics*, April, 1940) reviews the past classifications of ovarian tumours, demonstrates their inadequacies and proposes a new classification based on histogenetic principles. It is important to distinguish between retention cysts and true cystomata the result of neoplastic transformation. There are three types of retention cysts: follicular, *corpus luteum* and luteinized *corpus atreticum*, that is, lutein cysts. Of the true tumours there are two classes: first, ovariogenic tumours derived from ovarian tissue, and secondly, heterotopic tumours derived from tissue not normally present in the ovary. The first class is divided into granulosa cell tumours of varying types, fibromata either simple or luteinized, and myomata, angiomata and luteinomata, which arise from fœtal remnants of the tissue that physiologically develops into granulosa. The second group of true ovarian tumours develops from tissue which is not found in the normal ovary but is found elsewhere. There are two types. The first arises on the one hand by pathological differentiation of the cells of the surface epithelium forming serous cystoma resembling tubal epithelium, endometrioma resembling endometrium and pseudomucinous cystoma resembling cervical epithelium; and on the other hand from the mesenchymal core by an error in the sex chromosomes forming an arrhenblastoma producing male characteristics or a dysgerminoma which produces neutral sexual characteristics. The second type arises from displaced tissue. If this displacement occurs in fœtal life one of the following may be formed: a dermoid, an embryoma, a ganglioneuroma, a hypernephroma, a mesonephroma or a Brenner's tumour resembling urogenital epithelium. If the tissue is displaced in adult life either an endometrioma occurs by implantation or by metastases of malignant tumours an ovarian tumour is formed—for example, a Krukenberg carcinoma.

Formation of Artificial Vagina.

EUGENE P. STEINMETZ (*The Western Journal of Surgery, Obstetrics and Gynecology*, March, 1940) discusses congenital absence of the vagina with the presence of a rudimentary solid uterus. It is customary to find fairly well-developed external genitalia, though often somewhat rudimentary in type. The clitoris and *labia minora* are usually quite normal, while at the site of the vaginal orifice there is merely a dimple or a depression one to two centimetres deep. There is a connective tissue body isolating the rectum from the urethra and bladder. Dissection is easy in this connective tissue up to the pouch of Douglas. As there is occasionally an anomaly of the renal tract, it is wise before operation to locate the ureters. Engstad estimates that absence of the vagina occurs about once in 5,000 cases. The condition was first mentioned by Columbus in 1572, and Dupuytren in 1817 made the first known attempt to form an artificial vagina. In 1872 Heppner was the first to attempt to line the artificial pouch with skin grafts, and in 1897 Gersuny used mucous membrane of the rectum for this purpose. Baldwin in 1907 used a loop of small bowel as the lining. Mori in Japan about the same time used a single tube of ileum. From 1915 to 1920 Graves revived and improved the old skin flap method, and in 1927 Frank and Geist developed the modern tube or satchel-handle operation involving eighteen weeks' stay in hospital. Kerschner and Wagner in 1930 used Thiersch graft procedure, which afforded very satisfactory results, provided efficient post-operative dilatation of the new canal was maintained. In 1938 Robert T. Frank described the formation of an artificial vagina without operation, and the author followed this method of mechanical invagination. A narrow "Pyrex" tube, five-sixteenths of an inch in diameter and three inches in length, is firmly pressed into the hymenal region and the patient continues the treatment for half an hour three times a day for a week. Following this, tubes of varying sizes are used and the pressure is maintained overnight. In the author's case there was some slight erosion and granulation at the pressure point in the perineum. The patient continued to use the applicator for five months. She then married. Six weeks later she reported that coitus had been frequent and completely satisfactory to both, and that she had not needed the applicator. Examination revealed an artificial vagina seven centimetres deep, lined with soft resilient mucous membrane. The author concludes that the method is simple and satisfactory.

Carcinoma of the Body of the Uterus.

E. HELD (*Monatsschrift für Geburtshülfe und Gynäkologie*, Volume CX, Numbers 4 and 5, 1940) describes a method of applying radium in cases of corporeal carcinoma. It is applied in a series of tubes, six to fifteen in all, each containing 10 millicuries of radium element. The tubes are stitched in line like a rosary to a strip of gauze which is packed into the uterine cavity. Further radium is applied to the vagina by means of a colpostat. The radium is left *in situ* for twenty to forty-eight hours and the application is repeated after two or three weeks. The total average amount of radium used in the course of treatment is forty to fifty millicuries. Local inflammatory complications were not observed and no serious bladder lesions were noted.

The Treatment of Spontaneous, Threatened or Habitual Abortion.

C. G. COLLINS, J. C. WEED AND J. H. COLLINS (*Surgery, Gynecology and Obstetrics*, April, 1940) quote Shute to support their contention that the risk of the fœtus being malformed after a successfully treated threatened abortion is more theoretical than real. The authors used Shute's test for estimating the antiproteolytic power of blood serum from normal non-pregnant women, normal pregnant women, habitual aborters and women with threatened abortion. Their results were so inconsistent and inconclusive that they abandoned the test. They point out that there is no single substance that will consistently prevent spontaneous or habitual abortion and that a combination of substances of proved value will give the highest percentage of results. Accordingly they use wheat germ oil in all cases, with progestin or the anterior

pituitary-like hormone, and sometimes thyreoid extract. In this series the diagnosis of threatened abortion depended on the presence of cramping pain in the lower part of the abdomen persisting for longer than twenty-four hours, or uterine bleeding, whether only "spotting" or in larger quantities. The authors claim that with these conditions 87% of patients were saved from abortion. Cases were classified as habitual abortion if the patient had had two or more spontaneous abortions or in the immediately previous pregnancy had had signs of abortion. Eight out of twelve patients did not abort in the pregnancy under observation.

Cervico-Vaginal Fistula.

H. HELLENDALL (*Monatsschrift für Geburtshülfe und Gynäkologie*, Volume CX, Numbers 4 and 5, 1940) discusses the ætiology of cervico-vaginal fistula and states that in many cases it has followed attempts at criminal abortion. He describes in detail the reasons which he advanced in a criminal trial to support this conclusion. The literature is exhaustively analysed, and he concludes that the criminal ætiology must be granted precedence over the theory of spontaneous rupture in such cases.

OBSTETRICS.

Post-Partum Sterilization.

H. P. HEWITT AND J. R. WHITLEY (*American Journal of Obstetrics and Gynecology*, April, 1940) report 100 cases of tubal ligation within one hour of delivery. Skajaa, of Switzerland, and Adair have published reports of similar proceedings. All the patients were selected from those who had spontaneous deliveries. An incision one and a half inches long was used. The tubes were picked up at about the middle third and a large loop was crushed in three places. Each crushed place was tied with a linen suture inserted through the mesosalpinx. There was no mortality amongst the hundred patients. One patient developed pyelocystitis and one patient puerperal sepsis; this made a 2% morbidity. The authors come to the conclusion that the best time is one hour after delivery and that it is a safe procedure in selected cases.

Comparative Statistics of Maternal and Fœtal Mortality.

M. ROTHBAUM (*Monatsschrift für Geburtshülfe und Gynäkologie*, Volume CX, Numbers 2 and 3, 1940) has compared the maternal and fœtal results at the Zurich clinic over two periods, 1913 and 1936. The cases were divided into groups comprising young and elderly *primiparæ* and *multiparæ* and also into spontaneous and operative deliveries. There were fewer *primiparæ* over thirty in the earlier period, though operative interference in both groups was about equal. Such procedures are now more frequent with *primiparæ*, but lessened with parous patients. The incidence of Cæsarean section had greatly increased from 9 in 2,500 births in 1913 to 61 in an equal number of deliveries in 1936. While breech manipulations and manual removal of the placenta are more common, forceps application remains about the same, and internal version and craniotomy are much rarer. Maternal mortality among spontaneous deliveries amounts to 0·2%, compared with 3·8% in previous years. A noteworthy point was the increase in deaths among *primiparæ*, especially among those over thirty, as compared with *multiparæ*. The infant mortality shows a remarkable reduction from 8·1% to 4·7% for stillbirths and neonatal deaths.

The Lead Nipple Shield.

K. G. DODGE, in an editorial in the *American Journal of Obstetrics and Gynecology*, May, 1940, discusses the lead nipple shield. She quotes the two cases of lead encephalitis quoted by Wilcox and Coffey. A recent fatal case was reported and a review of the literature has been made by Bass and Blumenthal. It was believed that the lead lactate was sufficiently removed by well washing the nipple. However, large amounts of lead have been recovered from the milk of mothers using these shields. In all cases reported the shields were used for several months. Whether the amount ingested in a few days is harmful is open to question; but Bass and Blumenthal believe that many cases of unexplained anæmia, colic and convulsions occurring later in infancy may be due to lead in the body. These shields, with the accompanying literature, which states that they are "in no way likely to be injurious to the infant", are now forbidden to be sold in New York State and they have been banned for interstate commerce.

Neutral Diet and Hydration in the Toxæmias of Pregnancy.

R. R. DE ALVAREZ (*American Journal of Obstetrics and Gynecology*, March, 1940) discusses the treatment of 435 patients suffering from the toxæmias of pregnancy. For purposes of comparison the series was divided into two groups. The first group covered the period from 1901 to 1931 and the second group from 1931 to 1938. By this means it was possible to gain an idea of any improvement in treatment, as in the early years radical treatment was more common. It was noted that the highest maternal mortality was coincident with the high rate of Cæsarean section. The death rate for eclampsia was 20% in the earlier years and half that figure in the later years. During the past six years what the authors term hydration has dominated their medical management of toxæmias. It consists of rest, sedation, the administration of a neutral diet, of ammonium chloride and of an abundance of fluids. The food is prepared and served without salt.

Transcervical Cæsarean Section with Peritoneal Exclusion and Bladder Mobilization.

E. F. SMITH (*American Journal of Obstetrics and Gynecology*, May, 1940) describes a technique for the exclusion of the peritoneal cavity in the performance of a low Cæsarean section. The illustrations show how the bladder is freed and the peritoneum opened and then resealed in such a manner as to leave ample room for incision of the uterus and delivery of the child. The author claims that the technique affords a more roomy elastic area than any of the other exclusive operations, that it precludes the formation of post-operative adhesive bands, and that it restricts the operation to the well-oriented lower part of the peritoneal cavity, yet provides complete peritoneal exclusion.

Premature Infant Mortality.

R. M. GRIER AND H. O. LUSSKY (*American Journal of Obstetrics and Gynecology*, April, 1940) give a statistical study of factors influencing mortality in 453 infants weighing less than 2,500 grammes. They found that this group accounted for 65% of the total neonatal deaths. A graph shows the mortality curve for intrauterine age at birth and that for weight as nearly parallel. The authors found that the death rate for males was practically 50% greater than for females. The length of labour and the type of delivery, except that of breech extraction, seemed to have little effect on mortality. Morbidity, which was found in 37% of the mothers, had a definite effect on the infant mortality, being most pronounced in accidents of labour and hydramnios and least in toxæmia. The greatest number died in the first twelve hours, and the authors consider that this, the most critical time, should receive more study.

Naegele Pelvis.

R. IMBACH (*Monatsschrift für Geburtshülfe und Gynäkologie*, Volume CX, Numbers 4 and 5, 1940) describes the delivery of a young *primigravida* with an obliquely contracted pelvis of the Naegele variety. Antenatal supervision had revealed no abnormality, the external measurements were normal and the presentation was right occipito-anterior. Pains were weak until rupture of the membranes. Eight hours later the head was still mainly above the brim and the os was half dilated. An X-ray picture was taken and the cause of delay discovered. Labour was terminated by section. The case illustrated that external pelvimetry was not sufficient to diagnose the complication and that X-ray examination was essential in such cases.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

III.

ÆTIOLOGY: PHYSICAL FACTORS.

IN the assessment of physical factors in the causation of mental disease regard must be had to the personality of the patient, in order to determine the extent to which symptoms may be of recent development, or how far they are exaggerations of preexisting mental traits. A physical factor acts through impairing the efficiency of the physical mechanism underlying "mind" and here we may distinguish according to the Jacksonian scheme those negative symptoms which arise from the impairment of some function, for example, defective concentration or loss of memory, and those of a positive kind, due to release or decontrol, such as delusion formation and emotional instability. Moreover, and this applies more particularly to the neuroses, we must allow for psychological reactions to physical disease. The natural worrier may develop an anxiety neurosis in response to threatened or actual cardiac disease. There is, of course, a wide range of variation in the capacity to withstand physical insults, such as trauma, intoxication or disease. With a sufficiently intense infection the most stable individual is likely to become delirious, while at the other end of the scale the mental reaction may seem out of all proportion to the intensity of the noxious agent. The typical reaction to a physical factor is some degree of impaired comprehension (delirium and confusion) with loss of control over affective and conative responses. Such an acute organic reaction is seen, for example, in *delirium tremens*. Less frequently the acute reaction assumes the form of an acute mania or of a dull, confused, depressive state. In the less acute and more prolonged reactions there occur irritability, suspicions and delusions, sometimes hallucinatory depression. In a third category and with a still more prolonged course are cases with dulness, stupor and the confabulatory confusional syndrome of Korsakoff. Finally, actual destruction of neurones will be responsible for dementia. These organic reactions really show very little specificity with regard to separate physical factors. The mental complications of *diabetes mellitus*, uræmia, myocardial failure, anæmia and childbirth may assume any of these varieties. The nature of the mental response to physical disease depends essentially on the personality of the patient and the intensity and duration of the physical factor.

Fatigue.

Physical and mental exertion cannot be considered of itself a cause of any but a mild and transitory mental impairment (neurasthenic or confusional syndrome) unless performed over a long period and leading to decline in physical health. Usually the overwork complained of has been work against the grain or performed under emotional stress from some other cause. Hence the importance of inquiring into possible sources of worry and mental conflict whenever a person of reasonably stable mentality develops neurasthenic symptoms in the absence of physical disease. Improvements in the physical standards of living, important as they are in promoting physical health, leave untouched the multitude of psychological stresses which contribute towards neurosis; and wherever there is emotional stress there is fatigue, so that this factor, while it is less readily definable than many others, really appears to be one of the most common. Loss of sleep, of appetite and of weight may precede subjective and objective failure of mental efficiency and loss of emotional control. The tired child becomes irritable and emotional, and the tired adult reacts in much the same way. The part played by fatigue in any given case can be determined only after consideration of all the circumstances, including the patient's previous reactions to similar stresses. The fatigability complained of may develop without any special change in the patient's circumstances, in which case we may suspect that it is an early manifestation of melancholia, schizophrenia or of the dementia of a later age period. Occasionally a patient will admit that the nature and amount of the demands made upon him have in no wise changed, but that his capacity to cope with them has been failing. In other cases it may not always be wise to disabuse the patient's or his friends' minds of the fiction that he has been a slave to duty. It must be remembered that an actual increase in energy output may be a manifestation of a manic or an anxiety state.

Focal Sepsis.

The eradication of septic foci from teeth, nasopharynx, nasal sinuses, gastro-intestinal and genito-urinary tracts (especially the *cervix uteri* and prostate) has been claimed to promote materially recovery from the psychoses. On the other hand the general body of opinion amongst psychiatrists may be said to be conservative. Observers who have compared series of patients examined and treated by the methods of Cotton and Graves with those handled on well-established psychiatric principles, find no evidence of any quicker or better recovery rate in manic depressives and schizophrenics following tonsillectomy, antrotomy and other surgical procedures. More recently, of course, insulin and "Cardiazol" shock therapy have produced more startling results than any other treatments.

When any septic focus is discovered in a patient with mental symptoms the physician may fairly ask himself: "Would I advise this operation if the patient were free from mental symptoms?" For example, a man of previously stable mentality begins to feel "run down", out of sorts and easily fatigued and perhaps also has "rheumaticky" pains. No significant psychological factors are found and the physical examination reveals nothing beyond apical sepsis in, say, two or three teeth. One might expect that extraction in such a case would relieve not only the pains but also the general feeling of ill health. On the other hand a patient with melancholia or schizophrenia with the same physical findings would be most unlikely to derive any benefit from the removal of the septic foci. Especially in the neuroses is there a danger of attaching too much importance to mild sepsis and other disease, to the neglect of psychological factors, with disappointment to both patient and physician. But in delirious and confusional states every effort should be made to ascertain and treat any possible toxic infective conditions. Such a patient is unable to report and describe his symptoms like a mentally normal person, so that reliance must be placed on the general physical examination, temperature, pulse, urinalysis *et cetera*, together with such special investigations (for example, X-ray examination of nasal sinuses) as may seem indicated.

Trauma.

The following is a modified and condensed version of Adolf Meyer's classification of mental reactions to trauma:

1. The mental symptoms of concussion and cerebral compression and irritation.

2. Post-traumatic deliria. (*a*) Resulting from toxic-infective complications of trauma. (*b*) Due to "shock" as may occur also after operations. (*c*) Delirious phase intervening between emergence from coma and recovery. (*d*) Prolonged delirious-confusional phase with confabulations (Korsakoff's syndrome) with recovery even after months.

3. Post-traumatic constitutional changes. (*a*) Undue susceptibility to effects of alcohol, infective conditions and to physical and mental exertion. Under this heading we may include the syndrome described by Friedmann of

vasomotor and emotional instability, also termed the "explosive diathesis" of Kaplan. (*b*) Epileptiform developments including episodic memory lapses, fugues and other breaks in consciousness. (*c*) Paranoid developments.

4. Defect states. (*a*) Focal, such as aphasia. (*b*) General.

5. Psychoses such as mania, melancholia and schizophrenia and general paralysis following directly upon cerebral trauma, which acts only as a contributing factor.

6. Psychoses following upon trauma to regions of the body other than the head.

7. Neurotic reactions to physical injury, which as in the case of conditions mentioned in "5" is usually only a contributing factor.

Climate.

Adaptation to marked changes in temperature and humidity have, of course, their repercussions on mental efficiency. In a tropical country many new stresses have to be met. The debilitating effects of tropical diseases and of unsuitable diet need only be mentioned here. In the production of neurosis, so-called tropical neurasthenia, factors of a more psychological order play a greater part than physical disease. The changed mode of life, possibly isolation either complete or from contact with the patient's own race, nostalgia and the loss of the comforts of civilized life must all be considered. The monotony of the seasons and the unvarying length of days is depressing to many people living near the Equator. But above all, lack of moderation in alcohol and food, too little exercise, and the added demoralization of venery lead to impairment in both physical and mental health. Life in the tropics must be disciplined to be healthy.

Endocrine Imbalance.

The endocrine glands, the autonomic system and its centres in the mid-brain and hypothalamus work together in a system controlling the internal relationships of the body. The abnormal functioning of any one member may have its repercussions on the other components of the system, so that it may be very difficult to determine which is primarily at fault, and still more difficult to restore the balance of power by an appropriate remedy. In a consideration of the mental symptoms which may be associated with an endocrine disorder, regard must be had to the earlier personality of the patient and his mental history, with special reference to cyclothymic manifestations, which as likely as not occur independently of endocrine disturbances.

Thyreoid Gland.—It is hardly necessary to deal with cretinism and myxœdema in detail. Too much reliance should not be placed on a basal metabolic rate which is only some 10% or 15% below normal, since patients suffering from exhaustion (neurasthenia), melancholia and schizophrenia may give such a reading without other evidence of hypothyreoidism and without reacting favourably to thyreoid extract. Hypothyreoidism is in fact infrequent in the neuroses and psychoses. Nevertheless thyreoid deficiency may be responsible for the development of mental sluggishness at any age and in the presence of any suspicious physical signs the therapeutic test is always worth while, provided that it is carried out critically. Phases of excitement have also been ascribed to thyreoid deficiency.

Hyperthyreoid individuals usually display some emotional instability and may in fact present many of the features of an anxiety neurosis. Loss of weight, constant tremor and tachycardia, frequent vomiting and diarrhœa and a raised basal metabolic rate point to endocrine disorder rather than to a neurosis. In the milder forms, in the presence of only one or two suspicious symptoms and with possible psychogenic factors, decision as to the importance of endocrine as against other factors must for a time remain in doubt. Delirium and confusion may develop in thyreotoxicosis, although myocardial failure often contributes to the mental complications.

Pituitary Gland.—Under-activity of the anterior portion of the pituitary gland is responsible for delay in sexual and skeletal development and for psychic immaturity and lack of energy. Over-activity gives rise to gigantism or acromegaly according to the age at which the glandular dystrophy occurs. These changes may be associated with some increase of mental vigour at first, and during adolescence a failure to find a satisfactory outlet for this excessive energy may lead to delinquency. At a later stage the mental state is one of placidity, anergia or depression. The situational and functional relationships of the pituitary to the hypothalamus make it difficult to determine which may be responsible for emotional instability in suspected disease of these regions. The hypothalamus is closely concerned with the mechanism of emotional expression, and patients in whom this region has been affected by tumours or by disease, such as encephalitis, have exhibited outbursts similar to the "sham rage" observed in laboratory animals.

Adrenal Gland.—Lethargy, fatigability, muscular weakness and depression ("neurasthenic syndrome") occur with failure of the adrenal medullary secretion. Hyperplasia of the cortex is associated with virilism (adrenogenital syndrome), giving rise to hairiness and other masculine qualities in women, while in adolescent males there is sexual precocity. Most of the male patients are mentally deficient and display anti-social propensities. Delirium, convulsions and coma may be terminal features of adrenal failure.

Pancreas.—Irritability and depression are common mental accompaniments of *diabetes mellitus*. Some persons of an excitable temperament seem readily to use up their carbohydrate reserves and suffer exhaustion, faintness and headaches. The symptoms of this so-called glycopenic syndrome quickly disappear after the ingestion of readily assimilable carbohydrate. Confusion and sometimes great mental excitement may occur with any considerable reduction in the amount of blood sugar in the course of treatment by insulin and are constant features of the insulin shock therapy of the psychoses.

Gonads.—Hypogonadism is associated with physical and mental infantilism. Since the pituitary produces a gonadotropic secretion, sexual immaturity can rarely be ascribed to testicular or ovarian defect alone. Castration leads to a reduction in energy and aggressiveness. The vasomotor instability of the menopause is ascribed to oversecretion of prolan. How far emotional instability at this period can be referred to endocrine imbalance is a question to be decided in each individual case, since various other physical and psychological factors must be taken into consideration. Menopausal vasomotor instability may be relieved by the administration of the œstrogenic hormone, large doses of which have also been given with success in the depressive psychoses of this period. The changes in the testes and ovaries described by Mott in cases of *dementia præcox* have not been substantiated by other workers, so that the pathology of this condition still remains in doubt. Such pathological changes as can be demonstrated are probably incidental to rather than causative of the psychosis. Gross endocrine disorders are rarely seen in mental hospitals, but are more common in institutions for the congenitally defective.

The thymus and the pineal both normally cease to function as the gonads reach maturity. Tumours in or near the pineal cause under-activity of that gland and hence an absence of the normal restraining influence on sexual development. In children such tumours give rise to precocious mental and physical development (infant Hercules).

Parathyreoid Gland.—Confusion, excitement and depression are mental accompaniments of severe tetany due to defect of the parathyreoid gland.

W. S. DAWSON, M.D., F.R.C.P.,
Professor of Psychiatry, University of Sydney; Honorary Psychiatrist, Royal Prince Alfred Hospital.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 30, 1940, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, PROFESSOR W. K. INGLIS, the President, in the chair.

Cerebro-Spinal Fever.

DR. W. L. CALOV read a paper entitled "Cerebro-Spinal Meningitis" (see page 51).

DR. E. F. THOMSON said that cerebro-spinal fever was an infectious disease characterized by the suppurative inflammation of the meninges of the brain and spinal cord, particularly the meninges at the base of the brain. The disease might be either acute or chronic, although the chronic form was very rare. The disease occurred in both epidemic and sporadic form. The causal organism was the *Neisseria meningitidis*, commonly known as the meningococcus. The meningococcus belonged to that group of bacteria known as neisseria, the members of which were *Neisseria catarrhalis*, *Neisseria pharyngis*, *Neisseria meningitidis*, *Neisseria gonorrhœa* and *Neisseria flavescens*. Microscopically, these organisms were identical and appeared as Gram-negative, bean-shaped diplococci. They differed, however, in their cultural, biochemical and serological reactions, and could be readily distinguished by these means.

Dr. Thomson went on to say that the meningococcus was a strict parasite, and its normal habitat was in the naso-pharynx of man. Direct smears and cultures from the naso-pharynx had demonstrated the presence of the organism. It was difficult to cultivate, and on primary isolation required accessory growth factors, such as were present in blood, serum, milk and other animal extracts and certain vegetable extracts; hence such media as serum agar or blood agar were always used for primary isolation. After several subcultures on such media it might be possible to obtain the growth of the organism on more simple media; but the vitality of the organism under such conditions was uncertain. The meningococcal colony was generally a smooth lenticular colony; but the appearance tended to differ somewhat with the various types of meningococcus; Types I and III gave rough colonies, while Types II and IV gave smooth colonies. The meningococcus formed a very active autolysin, which destroyed the organism in cultures more than a few hours old. It was probable that this autolysin played an important part in the causation of the pathological lesion. The organism survived best at a pH of 7·4 to 7·6; the optimum temperature for growth was 37° C., and little or no growth occurred below 30° C. It was a strict aerobe. It was not resistant to inimical agencies. When dried and kept at room temperature it died in less than three hours. It was killed by moist heat at 55° in less than five minutes. A 1% solution of phenol killed it in one minute, and a 0·1% solution of mercuric chloride killed it almost instantaneously. It produced acid in glucose and maltose. The organism had a definite antigenic structure and had been divided into various groups by agglutination and absorption tests. There had been some argument as to the number of different types of meningococcus in existence; some groups of workers recognized two types, while others recognized four. Gordon and Murray recognized Types I, II, III and IV; Griffiths recognized Types I and II, his Type I corresponding with Gordon and Murray's Types I and III and his Type II with Gordon and Murray's Types II and IV.

Dr. Thomson went on to say that the meningococcus was pathogenic to man, and experimentally it was pathogenic to mice, guinea-pigs and rabbits if injected intraperitoneally in fairly large doses. As he had already said, the organism had its natural habitat in the naso-pharynx in man, and could be present in the naso-pharynx without causing any pathological effect. It was this fact which gave rise to the carrier problem in cerebro-spinal fever. There was now a general agreement that the naso-pharynx was the portal of entry, but there was still some argument as to the exact route by which the organism reached the central nervous system. One school of thought favoured direct extension from the nose through the cribriform plate, either by extension along the sheaths of the branches of the olfactory nerve or by the lymphatics. There was no question but that there was a close relationship between the subarachnoid space and the nose. It was also possible that there might be extension from the nasal sinuses. The other school of thought favoured a blood-borne infection, and that view was supported by the fact that in the early stages of the disease the organism could in some cases be recovered from the blood. Some of the clinical features, such as the rash, rather suggested an initial septicæmia.

Dr. Thomson went on to say that the probable sequence of events was that the organism reached the naso-pharynx by air-borne infection. Here it either gave rise to no trouble at all or set up a rhino-pharyngitis. If the organism progressed no farther than the naso-pharynx, then the patient became a carrier; when meningitis developed, the organism passed from the naso-pharynx to the meninges, most probably by the blood stream, although direct extension through the cribriform plate could not be excluded. The organism finally became localized in the meninges and exerted its pathogenic effects. The possibility that the disease was primarily a septicæmia and that the organism reached the central nervous system by the choroid plexus was supported by the fact that in some cases of twenty-four to forty-eight hours' duration culture from the blood during life, culture from the heart blood at autopsy and culture from the ventricular fluid had yielded the meningococcus, while the meninges appeared to be normal.

Dr. Thomson then referred to the morbid anatomy. He said that the main pathogenic changes were seen in the brain and spinal cord, although these changes varied with the duration of the disease. In fulminating cases, in which death occurred in forty-eight hours, there might be little to see. In a typical case of cerebro-spinal fever, when the skull cap was removed the dura was seen to be greatly congested, intense and bulging. When the dura was incised, a yellowish or purulent exudate was found covering the surface of the brain. This was most pronounced at the base of the brain, although in advanced cases there was also considerable exudate over the vertex. The brain was swollen and œdematous and the convolutions were flattened. The vessels at the surface of the brain were intensely engorged. The purulent exudate tended to follow the line of the surface vessels.

When the brain was opened the ventricles were found to be moderately dilated and filled with purulent cerebro-spinal fluid. The choroid plexus was very congested and had a dull, greyish appearance. Similar changes were found in the spinal cord, although the changes were more often than not confined to the posterior aspect of the cord. Microscopically the picture was that of an acute inflammation with pus formation. The other organs of the body showed the usual evidence of septicæmia, such as congestion and cloudy swelling. The naso-pharynx was much congested. Meningococcal endocarditis, with or without meningitis, had been described.

Referring to the laboratory diagnosis, Dr. Thomson said that attempts at culture from the blood were not always made; it was advisable, however, to attempt a culture in the early stages of the disease, especially when the purpuric rash was present. It was said that in 25% of cases a positive blood culture was obtained. Naso-pharyngeal swabbing should be carried out; in an endeavour to discover the presence of the meningococcus in the naso-pharynx West's naso-pharyngeal swab should be used. The third aspect of laboratory diagnosis was the examination of the cerebro-spinal fluid. In most cases of cerebro-spinal fever the fluid had a characteristic appearance. It was increased in amount and was obtained under considerable pressure. It was turbid, the turbidity being uniform. There was a great increase in the cell content of the fluid, up to as many as 3,000 per cubic millimetre. The cells were of the polymorphonuclear type. Examination of a stained smear of the deposit after the cerebro-spinal fluid had been centrifuged revealed as a rule the typical bean-shaped Gram-negative diplococci in an intracellular position. The organisms were not always found, however, and might be absent even in acute cases. Culture of the cerebro-spinal fluid usually resulted in a pure growth of meningococci. Sometimes an attempt at direct culture from the fluid yielded no result, and it was always wise to incubate a small portion of the fluid at 37° C. for twelve hours before the attempt at culture was made. If this was done, a culture would almost always be obtained. Chemically there were a great increase in the protein content of the fluid and a slight diminution of the chloride content (rarely below 600 milligrammes *per centum*); there was an almost complete absence of sugar. The Lange gold chloride reaction usually produced a typical curve with a rise at the right-hand end. Dr. Thomson referred particularly to the fact that it might prove impossible to obtain a culture after the use of chemotherapeutic agents. An absence of culture should not rule out the diagnosis of cerebro-spinal meningitis, and the drug should not be withheld in order to obtain the diagnosis.

Dr. Thomson then went on to speak of the carrier problem and prophylaxis. He said that there was no doubt that the disease was spread by carriers, and that was of importance both in civil life and in time of war. The outbreaks of cerebro-spinal fever in military camps were well known: there had been many examples during the Great War of

1914-1918. Similar outbreaks would occur during the present war, and had indeed already done so in some of the military camps. It was interesting to note that at the beginning of 1940 there had been a widespread and very severe epidemic of cerebro-spinal fever in Great Britain; 586 cases had occurred between February 17 and 24. No comparable outbreak had taken place since the first winter of the last war.

It was true that it was unusual for a patient suffering from cerebro-spinal fever to affect another person, and the disease was spread almost entirely by carriers. Under normal conditions the meningococcus would be found in a normal proportion of apparently healthy persons, and as a conservative estimate it could be said that in non-epidemic times the meningococcus was present in 2% to 8% of healthy civilians. During epidemic periods the carrier rate rose, and Glover had shown that in the presence of severe overcrowding the carrier rate might rise from 20% to 60%. A carrier rate of 20% was a sign of danger, and usually preceded an epidemic. It was interesting to note, however, that although these facts were substantially true, the actual carrier rate was not always a guide to the onset of an epidemic. For instance, it had been found that at the Chatham Naval Hospital there were 11 cases of cerebro-spinal fever and a carrier rate of 13%; in the following season, however, there was a carrier rate of 54% and no case of cerebro-spinal fever occurred; moreover, the organism changed from Type II to Type III. During a similar period in the naval hospital at Portsmouth there were six cases of cerebro-spinal fever with a carrier rate of only 5%. Hence it was fairly clear that the occurrence of an epidemic of the disease depended not only on the number of carriers, but also on the virulence and the type of the organism.

With regard to carriers, Glover had shown that in the last war the carrier rate of so-called non-contacts was equal to that of so-called contacts among those living under similar conditions in a community in which there were cases of cerebro-spinal fever. Clearance of the naso-pharynx by the mechanism of natural resistance in healthy subjects was commonly a matter of hours or days. A person from whose naso-pharynx meningococci were recovered by swabbing on one day might be found to harbour none on the next day, and *vice versa*. Further, the type of meningococcus present in the naso-pharynx was important; but it was almost impossible to tell on routine examination, even though the organism was typed, whether the particular organism was virulent in the sense of possessing the power to invade the blood stream and the meninges. Hence, as the Ministry of Health in Great Britain had pointed out, it was not so much the search for carriers that would stamp out an epidemic as the recognition and correction of other predisposing factors.

Nevertheless, the excellent work of Glover during the Great War still stood, and, as he had pointed out, severe overcrowding with a space of only one foot between the beds and at the same time obstruction of ventilation were very important factors, which determined the occurrence of an epidemic. The outstanding predisposing factors were thus overcrowding, insufficient ventilation, naso-pharyngeal catarrh, the recent introduction of susceptible persons, for example recruits, into the community, and the presence of highly virulent organisms. The control of the disease therefore depended upon six measures: (i) the provision of at least three feet of space between beds in sleeping quarters, especially those occupied by recruits, school children and other young people; (ii) the provision of some device for maintaining free ventilation while excluding light in at least a portion of the windows of dormitories, canteens, recreation and living rooms which were occupied after sunset; (iii) the avoidance of the introduction of "non-immunes" into the herd; batches of recruits or school children should be kept together for considerable periods without the constant addition of newcomers to their numbers; (iv) the control, as far as possible, of naso-pharyngeal catarrh (the inflammation accompanying naso-pharyngeal catarrh appeared to reduce the phagocytic activity available against the meningococcus and might aid its absorption into the blood stream; coughing and sneezing aided the dissemination of meningococci from throat to throat); (v) the early diagnosis of the condition (the chief symptoms were a papular or petechial rash, pyrexia, irritability, hyperæsthesia, pain and tenderness in the neck); (vi) early treatment by chemotherapy.

Dr. Thomson finally referred to the reduction in mortality rate that had occurred with the use of chemotherapy; with serum treatment the mortality rate was 50% and with chemotherapy it had been reduced to 8%. Dr. Thomson urged those present to read articles appearing in *The Lancet* of January 6, March 16 and March 23, 1940, and in *The British Medical Journal* of March 9 and March 23, 1940.

DR. E. H. M. STEPHEN drew attention to the fact that a rash was not an invariable accompaniment of cerebro-spinal fever. In fact it was such an uncommon occurrence that he could recall to mind only one instance of a well-developed typical rash, and had broadcast the news to the other members of the staff in order that such a unique opportunity should not be missed. In these circumstances the presence or absence of a rash was not of much assistance in making a diagnosis. A pronounced head retraction in the early days of an illness was in his experience suggestive of meningococcal meningitis. The success of cisternal puncture was mentioned in a case in which the pathologist had been unable to find the causal organisms in fluid obtained by lumbar puncture.

Dr. Stephen then mentioned a patient who had developed pituitary dystrophy as a sequela of meningococcal meningitis. Within a few months this boy had attained the weight of eight stone at the age of ten years, with typical appearance. The pituitary dystrophy had persisted until the age of about fifteen or sixteen years, and after that the boy became a particularly tall young man. According to his mother, he did brilliantly at school; after that he was employed in a Government department, in which, according to his mother, he did very well.

Dr. Stephen hoped that all would go well with patients suffering from cerebro-spinal meningitis, now that "M & B 693" was used in treatment. Previous to the introduction of this drug in treatment, prognosis in children under the age of six months had been definitely bad, and for those under the age of twelve years the prognosis was not much better. In conclusion Dr. Stephen expressed his appreciation of the two papers that had been presented.

DR. RICHMOND JEREMY said that he had two patients suffering from cerebro-spinal meningitis in his beds at the Prince Henry Hospital, and in each instance he had not seen the patient on admission to hospital. He saw them first three or four days afterwards, and both patients asked him when they could go home. One of them had been admitted to hospital in deep coma. Dr. Jeremy then referred to the occurrence of cerebro-spinal meningitis amongst soldiers. He said that treatment with sulphapyridine had so shortened the period of invalidism that patients seemed perfectly well at the end of a week. He wondered whether the military authorities would recognize the usually accepted period of convalescence or whether they would send the men back to duty when they appeared to have recovered.

DR. N. J. SYMINGTON said that a few patients were being sent to hospital from military camps with a diagnosis of cerebro-spinal meningitis and their illness turned out to be German measles. People seemed to put too much stress on a rash as a means of diagnosis, and it was thought to be one of the main features; but it was not. The patients to whom Dr. Symington referred had pain at the back of the neck because of enlarged tender glands, and this evidently gave the erroneous impression of neck rigidity.

DR. MARJORY LITTLE said that the identification of the meningococcus had been made much easier now that it was possible to use the special media employed for the cultivation of the gonococcus and the special dyes for differentiation of colonies. During the last war they had used transparent media on which the meningococcus grew quite well; but on this medium it was difficult to pick out different colonies for testing on the sugars, and in the case of carriers particularly the use of special dyes made the recognition of the meningococcus easier. Dr. Little said that she had not seen much of the disease lately, but during the last few weeks had examined the cerebro-spinal fluid from a baby, aged five weeks, who had contracted the disease. Her experience confirmed that of Dr. Thomson on the difficulty of finding the meningococcus in immediate spheres of cerebro-spinal fluid; but a heavy growth of meningococci had been obtained from the specimen on the second day. The baby was treated with sulphapyridine and in a few days was perfectly well.

DR. OLIVER LATHAM said that he supposed the introduction of "M & B 693" therapy had made the typing of meningococci unnecessary. At one time he had shared a laboratory with a Captain Caldwell, Royal Army Medical Corps, who was receiving specimens of spinal fluid, usually already plated and kept warm, from meningitic cases from all over Salisbury Plains. These had been remitted for confirmation and typing. Dr. Latham had already discussed with him the Bass Watkins rocking slide method for agglutinating formol suspensions of typhoid bacilli. Captain Caldwell applied his four types of serum to drops of the spinal fluid cultures on the one large sheet of glass and quite often was enabled to recognize the type of meningococcus involved and telephone the result in a few minutes.

PROFESSOR W. K. INGLIS expressed to those who had read the papers the appreciation and thanks of the meeting.

Dr. Thomson, in reply to Dr. Latham, said that his supposition was correct; there was no longer any need to bother typing meningococci. Dr. Thomson was interested in

Dr. Jeremy's question concerning soldiers who had made rapid recoveries and the attitude of the military authorities to the period of convalescence necessary. He wondered whether the men would be required to resume duty after a fortnight. An interesting point to him was that while there was this new drug, with which they appeared to be able to treat anything at any time, they were not sure of what would happen to the patients later on. Certain relapses occurred in gonorrhœa, which perhaps were not expected. While these cerebro-spinal meningitis patients recovered and seemed to be quite well in a fortnight, should they be watched for a longer period or should they be allowed to go about their ordinary work? That was a point to keep in mind. From his own experience Dr. Thomson did not know whether a rash was common or not; but articles in recent journals had certainly mentioned the presence of a rash as one of the symptoms of cerebro-spinal meningitis.

NOTICE.

THE Criminal Investigation Branch of Victoria advises that several bags of instruments have been stolen from unlocked motor cars in Melbourne in recent weeks, and seeks the cooperation of the medical profession in the prevention of such thefts and of the disposition of stolen property.

Members are asked to see that untended motor cars are left locked and, should they be offered instruments for purchase, except through reputable trade channels, to communicate at once with Dr. C. H. Dickson, Medical Secretary of the Victorian Branch.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the University of Adelaide on April 5, 1940.

Human Heredity.

A paper was presented by Dr. I. F. Phipps on "Recent Developments in the Field of Human Heredity". Dr. Phipps pointed out that the analysis of inheritance in man must be carried out by indirect methods, the usual and efficient method of controlled breeding being impracticable. He said that recent advances in our knowledge of human heredity had involved improvements in these indirect methods based on the application of new discoveries in applied mathematics and refinements in statistical analysis. The new developments had been concerned with sex linkage, the discovery of genes by the *Y* chromosome, mutation rates, allelomorphic genes for the blood group series *AB* and *MN*, the phenomenon of incomplete penetrance, and the fate of genes in a population under assumed conditions of survival.

Identical twins, which from the standpoint of heredity were the same individual in duplicate, permitted the effect of environment to be studied independently of heredity.

The study of chromosome behaviour in a particular individual was now possible as a result of the discovery of a method of culturing leucocytes whereby abundant cell divisions were produced.

The pitfalls involved in the political application (compulsory sterilization) of our knowledge of heredity in man were now better understood. The rapidly growing relationship between heredity and medicine suggested that the subject should have a more prominent place in the curriculum of medical students. The paper was illustrated with pedigree charts, two of which were collected in Adelaide.

Post-Graduate Work.

COURSE IN ANATOMY AND PHYSIOLOGY AT SYDNEY.

THE eleven weeks' course suitable for candidates for Part I of the examination for the degree of Master of Surgery which has been arranged by the New South Wales Post-Graduate Committee in Medicine, to be held at the University of Sydney, will begin on Tuesday, July 30, 1940. Those intending to take whole or part of the course are asked to forward their applications to the secretary of the committee, the Prince Henry Hospital, Little Bay, from whom copies of the programme are available. Medical officers of the defence forces who are on full-time service will be permitted to attend free of charge.

LIBRARY SEMINAR AT SYDNEY.

THE next library seminar and clinico-pathological conference arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology, will be held at the Prince Henry Hospital, Little Bay, on Monday, July 22, 1940. The subject is "Fluid Balance in Disease". A cordial invitation to be present is extended to all medical practitioners.

The Royal Australasian College of Physicians.

A MEETING of the Royal Australasian College of Physicians will be held in Sydney on Friday and Saturday, September 6 and 7, 1940. The programme will include a council meeting, general meeting and scientific sessions on the afternoon of Friday, September 6, and on the morning of Saturday, September 7.

On the evening of Friday, September 6, a joint scientific meeting of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons will be held. The subject for discussion will be "Post-Operative Thrombosis and Embolism", and the speakers will be Dr. J. G. Hayden and Mr. Balcombe Quick, of Melbourne.

The Acting Honorary Secretary will be glad to receive offers of scientific contributions to be given at the afternoon and morning sessions of September 6 and 7 from Fellows and Members of the College on or before August 10, 1940.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 124, of July 4, 1940.

NAVAL FORCES OF THE COMMONWEALTH

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Ante-dating Seniority.—The seniority of Surgeon Lieutenant Athol Herbert Robertson is ante-dated to 27th September, 1939.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Extension of Appointment.—The appointment of Surgeon Lieutenant-Commander Donald Dunbar Coutts is extended for a period of one year from 29th April, 1940.

Royal Australian Naval Volunteer Reserve.

Termination of Appointment.—The appointment of Surgeon Lieutenant (on probation) James Biggam Douglas Galbraith is terminated, dated 16th May, 1940.

AUSTRALIAN MILITARY FORCES.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captain—James Alexander Cran, 5th June, 1940.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps.

To be Captain (provisionally)—Stewart Lloyd Cameron, 23rd May, 1940. Captain G. E. Crowther ceases to be seconded and is transferred to the Reserve of Officers (A.A.M.C.), 27th March, 1940.

Australian Army Medical Corps Reserve.

To be Temporary Lieutenant-Colonel—Major W. Evans, M.C., 16th May, 1940. Captain R. L. Bellamy is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

To be Major (temporarily)—Thomas Rhodes Matson, 5th June, 1940. *To be Captains (provisionally)*—James Eric Clarke and Douglas John MacLeod Dunn and to be borne supernumerary to establishment pending absorption, 24th May, 1940; Robert Keith Edwards, 30th May, 1940; Patrick Neil O'Donnell and to be borne supernumerary to establishment pending absorption, 31st May, 1940. Captain H. S. Jacobs ceases to be seconded, 7th June, 1940.

Australian Army Medical Corps Reserve.

To be Major—Edwin Blomfield Gerard Riley, 30th May, 1940. *To be Honorary Captains*—Boyns Hedley Hocking, Percy Robert Stephens, Ronald William Sharp and James Hudson Paterson, 5th June, 1940.

Fourth Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Carl Hannaford Schafer and John Harold Petchell, 28th May, 1940, and 4th June, 1940, respectively.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Neville Goldsbrough Row, Roland Hodgson Nattrass and John Alexander Gollan, 4th June, 1940.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The following Flight Lieutenants are granted the temporary rank of Squadron Leader with pay of that rank, with effect from 1st June, 1940: N. H. Andrews, W. L. B. Stephens, C. J. N. Le Leu and E. H. Anderson.

Citizen Air Force.

Medical Branch.

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from the dates shown: Henry Arthur Sundstrup, M.Sc., M.B., Ch.M., 15th May, 1940; Barry Joseph Mulvany, M.B., B.S., Harold Adrian Fulton Rofe, M.B., B.S. 10th June, 1940; Roy Vernon Pridmore, B.Sc., M.B., B.S., 12th June, 1940; Ronald William Hazelton, M.B., B.S., D.O. (Oxon), D.O.M.S. (London); Desmond Lees Peate, M.B., B.S., 17th June, 1940; Don Cresswell Howle, M.B., B.S., 20th June, 1940; John Herbert Bilton, M.B., B.S., 24th June, 1940, and Maurice Morris, M.B., B.S., 29th June, 1940.

The following are granted commissions on probation with the rank of Flight Lieutenant and the acting rank of Squadron Leader with effect from 1st July, 1940: John Joseph Witton Flynn, M.C., B.A., M.B., Ch.M., and Thomas A'Beckett Travers, M.B., B.S., M.R.C.P. (London), D.O.M.S. (London).

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from 1st July, 1940: Paul Alban Benbow, B.D.S., Cecil Lloyd Harold Bott, Dip.D.S., Peter Eugene Breheny, M.B., B.S., John Cumming Burns, B.D.S., William Peter Harvey Dakin, M.B., B.S., Rupert John Dingle, B.D.Sc., L.D.Q., Harry Richard Hall, B.D.S., Eric John Gee, M.D.S., Frank Vivian Munro, M.B., B.S., Athol Frederick Blair Stone, Dip.D.S., and Alan Victor Ward, B.D.S., L.D.Q.

David Buick Skewes, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 8th July, 1940.

Squadron Leader W. D. Counsell is granted the temporary rank of Wing Commander with pay of that rank with effect from 1st June, 1940.

The following Flight Lieutenants are granted the temporary rank of Squadron Leader with pay of that rank with effect from 1st June, 1940: N. E. H. Box, S. F. Reid, Honorary Squadron Leaders I. G. McLean, L. B. Cox, C. G. H. Blackmore, H. S. Kirkland, B. A. Hunt, D. R. Gawler, C. A. Hembrow and R. L. T. Grant.—(Ex. Min. No. 38—Approved 3rd July, 1940.)

Reserve.

Medical Branch.

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from 1st July, 1940: William McLean Borland, M.B., B.S., Baden Randell Cooke, M.B., M.S., Henry Coverley Maling, M.B., B.S., F.R.C.S. (Edin.) and Percy Robert Stephens, L.D.Q.—(Ex. Min. No. 36—Approved 3rd July, 1940.)

THE SECOND AUSTRALIAN IMPERIAL FORCE.

THE following statement is published at the instance of the Director-General of Medical Services.

The formation of the Eighth Division, Second Australian Imperial Force, for service overseas will necessitate the staffing of the following medical services:

1 General hospital (1,200 beds) ..	31 medical officers.
1 General hospital (600 beds) ..	19 medical officers.
1 Casualty clearing station	8 medical officers.
3 Field ambulances	27 medical officers.
1 Field hygiene section	1 medical officer.
Regimental medical officers	22
Hospital ship	5 medical officers.

The total number of medical officers required is approximately 113. Of these, specialist appointments account for 42, made up as follows: surgeons, 17; physicians, 11; radiologists, 5; pathologists, 2; otologists, 2; anæsthetists, 3; ophthalmologists, 2. Many applications for service overseas have been made, and some appointments have been approved, but vacancies still exist.

Those medical practitioners who submitted their names for service overseas with the Sixth and Seventh Divisions will be considered available for future appointments unless word is received from them to the contrary.

Applications from both male and female medical practitioners for enlistment with the Australian Army Medical Corps for service at home or abroad should be made to the Deputy Director of Medical Services of each military district.

Honours.

BIRTHDAY HONOURS.

AMONG the birthday honours conferred by His Majesty the King on members of the Australian Navy, Army and Air Force is one granted to a medical practitioner from Victoria who is a combatant officer in the Eighth Division of the Australian Imperial Force. Brigadier Edmund Frank Lind, D.S.O., V.D., has been created a Commander of the Military Division of the Most Excellent Order of the British Empire. Warm congratulations are offered to Brigadier Lind.

Obituary.

VICTOR BERESFORD TAYLOR.

WE regret to announce the death of Dr. Victor Beresford Taylor, which occurred on July 7, 1940, at Brisbane, Queensland.

Books Received.

FORENSIC MEDICINE. A TEXTBOOK FOR STUDENTS AND PRACTITIONERS, by S. Smith, M.D., F.R.C.P., D.P.H., with an introduction by H. Littlejohn, F.R.C.S., F.R.S.E.; Seventh Edition; 1940. London: J. and A. Churchill Limited. Demy 8vo, pp. 696, with illustrations. Price: 25s. net.

ST. THOMAS'S HOSPITAL REPORTS: SECOND SERIES, VOLUME IV, edited by O. L. V. S. De Wesselow, C. M. Page, with the assistance of N. R. Barrett, J. St. C. Elkington and A. J. Wrigley; 1939. London: St. Thomas's Hospital. Royal 8vo, pp. 198, with illustrations. Price: 10s. net.

SHELL SHOCK IN FRANCE 1914-1918, BASED ON A WAR DIARY, by C. S. Myers, C.B.E., F.R.S.; 1940. Cambridge: The University Press; Melbourne: G. Jaboor. Crown 8vo, pp. 152. Price: 4s. 6d. net.

PHYSICAL TREATMENT BY MOVEMENT, MANIPULATION AND MASSAGE, by J. B. Mennell, M.A., M.D., B.C.; Fourth Edition; 1940. London: J. and A. Churchill Limited. Demy 8vo, pp. 682, with 281 illustrations, including 32 plates. Price: 25s. net.

TOMOGRAPHY, by J. B. McDougall, M.D., F.R.C.P., F.R.S.E.; 1940. London: H. K. Lewis and Company Limited. Demy 4to, pp. 73, with 110 illustrations. Price: 21s. net.

THE VIRUS, LIFE'S ENEMY, by K. M. Smith, F.R.S.; 1940. Cambridge: The University Press; Melbourne: G. Jaboor. Crown 8vo, pp. 188, with illustrations. Price: 7s. 6d. net.

THE ANATOMY OF THE FEMALE PELVIS, by F. A. Maguire, C.M.G., D.S.O., V.D., M.D., Ch.M., F.R.C.S., F.R.A.C.S., F.R.C.O.G.; Third Edition; 1940. Australia: Angus and Robertson. Demy 8vo, pp. 172, with illustrations. Price: 10s. 6d. net.

THE BRITISH ENCYCLOPÆDIA OF MEDICAL PRACTICE, INCLUDING MEDICINE, SURGERY, OBSTETRICS, GYNÆCOLOGY AND OTHER SPECIAL SUBJECTS. SURVEYS AND ABSTRACTS, 1939, under the general editorship of Sir Humphry Rolleston, Bt., G.C.V.O., K.C.B., M.D., D.Sc., D.C.L., LL.D.; Publishing Editor: A. Clark, L.M.S.S.A.; Sub-Editor: G. Faulkner, D.Sc.; 1940. London: Butterworth and Company (Publishers) Limited. Super royal 8vo, pp. 678, with illustrations. Price: 32s. 6d. net.

A SHORT PRACTICE OF MIDWIFERY FOR NURSES, by H. Jellett, B.A., M.D., F.R.C.P.I.; Eleventh Edition; 1940. London: J. and A. Churchill Limited. Crown 8vo, pp. 528, with illustrations.

YOU AND HEREDITY, by A. Scheinfeld, edited by J. B. S. Haldane; 1939. London: Chatto and Windus; Australia: Angus and Robertson. Demy 8vo, pp. 453, with illustrations. Price: 16s. net.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Burgess, John Sanday, M.B., B.S., 1940 (Univ. Sydney), Royal North Shore Hospital of Sydney, St. Leonards.
Read, George, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Epps, William Glover Howie, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.
Horan, Francis James, M.B., B.S., 1933 (Univ. Sydney), St. George Street, Mungindi.
Mackay, Robert Wadie Gordon, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Paterson, Lucie Doris, M.B., B.S., 1938 (Univ. Sydney), "Crawney", Murrurundi.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Ferguson, William Frederick, M.B., B.S., 1940 (Univ. Melbourne), "Mareeba" Babies' Hospital.
Woods, Charles William Griffin, M.B., B.S., 1939 (Univ. Adelaide), Kilkenny.

The undermentioned has been elected a member of the South Australian Branch of the British Medical Association:

Good, Henry Arthur, M.B., B.S., 1939 (Univ. Adelaide), Bordertown.

Diary for the Month.

JULY 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 24.—Victorian Branch, B.M.A.: Council.
JULY 25.—South Australian Branch, B.M.A.: Branch.
JULY 25.—New South Wales Branch, B.M.A.: Branch.
JULY 26.—Tasmanian Branch, B.M.A.: Council.
JULY 26.—Queensland Branch, B.M.A.: Council.
AUG. 1.—South Australian Branch, B.M.A.: Council.
AUG. 2.—Queensland Branch, B.M.A.: Branch.
AUG. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 7.—Victorian Branch, B.M.A.: Branch.
AUG. 7.—Western Australian Branch: Council.
AUG. 8.—Queensland Branch, B.M.A.: Council.
AUG. 13.—Tasmanian Branch, B.M.A.: Branch.
AUG. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 20.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Associated Medical Services Limited. All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Federated Mutual Medical Benefit Society. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

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